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Original Articles

PAGET'S DISEASE IN THE MAXILLA, MANDIBLE, AND PALATE

A Histologic Analysis

Irving Glickman, B.S., D.M.D.,* Boston, Mass.

ALTHOUGH microscopic changes in Paget's disease have been described in detail in the remainder of the skeletal system,[1-4] a thorough study of the changes in the bones of the jaws has not been reported. It was felt that such a study was needed for two reasons; first, to establish in detail the morphologic changes in the jaws in Paget's disease, and second, to supply information which might be applied in interpreting changes in these bones in conditions other than Paget's disease. This paper is devoted to a detailed description of the salient morphologic features observed in the maxilla, mandible, and palate when these bones are involved in Paget's disease. In addition, a comparison between the findings in these bones in Paget's disease and in non-Paget individuals of a similar age group, will be presented.

MATERIAL AND METHODS

The material for this study is derived from two adult male patients, aged 66 and 47 years. In one patient, the right maxilla was the only bone in which clinical and radiographic changes consistent with Paget's disease were observed. Sections were made from biopsy material from the involved area for microscopic study. In the other patient, in addition to the maxilla, mandible, and palate, the skull, vertebrae, sternum, and extremities presented characteristic clinical and radiographic changes. Serial sections were made of the palate, maxilla, and mandible of this patient. In both patients the jaws were edentulous.

All of the material was fixed in 10 per cent formalin, decalcified with an equal mixture of 45 per cent formic acid and 20 per cent sodium citrate, and embedded in paraffin.

OBSERVATIONS

A. Bone Marrow.—The marrow of the mandible, maxilla, and palate presents a variable picture. In each of these bones there are areas in which the marrow spaces are filled with a dense meshwork of connective tissue cells and fibrils. The connective tissue cells vary in shape from a thin elongated cytoplasm with a flattened, deeply staining nucleus, to a polyhedral shape with large rounded nucleus containing prominent chromatin granules. The predominance of such zones of connective tissue marrow is a striking feature. There are other areas in which the marrow is composed predominantly of fat cells (Fig. 3). In many zones, the marrow presents a mixture of varying proportions of connective tissue cells and fibers and fat cells. The distribution of the marrow contents is spotty in nature. In some instances one type of marrow blends with another

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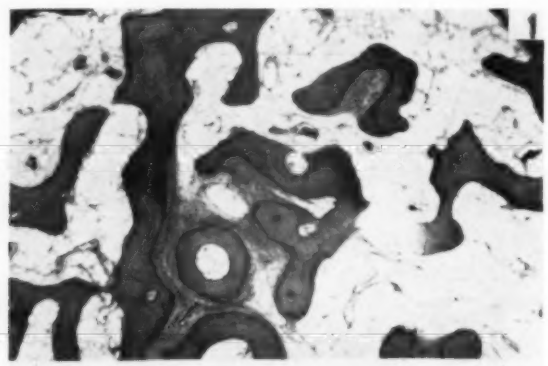
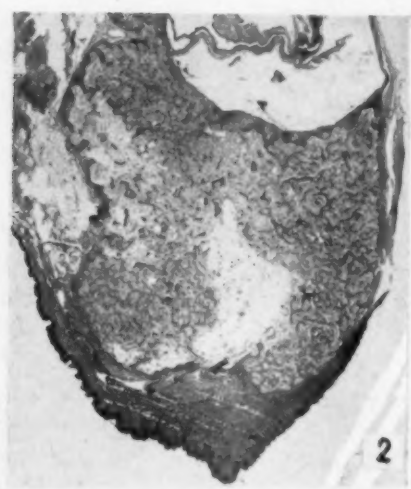
type adjacent to it, while, in others, a small zone of connective tissue marrow appears isolated and sharply demarcated from the surrounding zone of fat cells (Fig. 1). There is no apparent rationale to this patchy disposition of several types of marrow. In the mandible, it appears that the marrow nearest the crest of the ridge is predominantly connective tissue in nature, whereas,





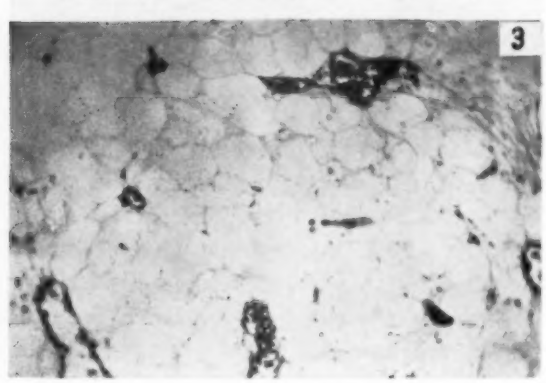


Fig. 1.—Palate. Isolated bone of connective tissue marrow sharply demarcated from surrounding fat marrow cells. Hematoxylin and eosin. Magnification ×24.

Fig. 2.—Left maxilla. Oral mucosa on the right covered with stratified squamous epithelium. Marrow is fibrous throughout with the exception of area immediately underlying the crest of the alveolar ridge. Hematoxylin and eosin. Magnification ×8.

Fig. 3.—Detail of Fig. 2. Area underlying crest of ridge comprised of marrow fat cells. Hematoxylin and eosin. Magnification ×142.

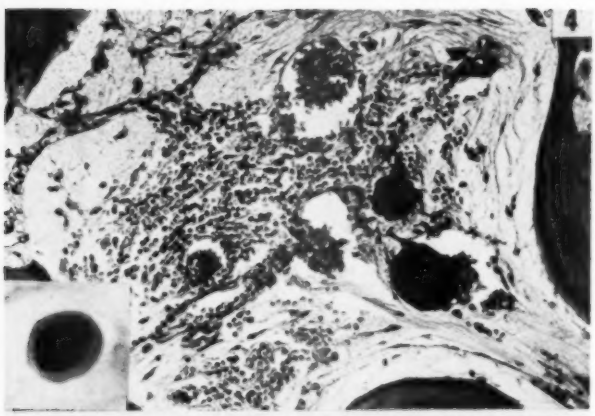
that deeper in the bone, less intimately connected with functional stress, is composed of fat cells. However, this relation of marrow content to functional stress is not a consistent finding. In the left maxilla directly subjacent to the subepithelial connective tissue of the crest of the ridge, a sharply demarcated zone of marrow comprised of large fat cells (Figs. 2 and 3) is seen surrounded by markedly fibrous marrow in areas less closely related to the zone of greatest function at the crest of the ridge.

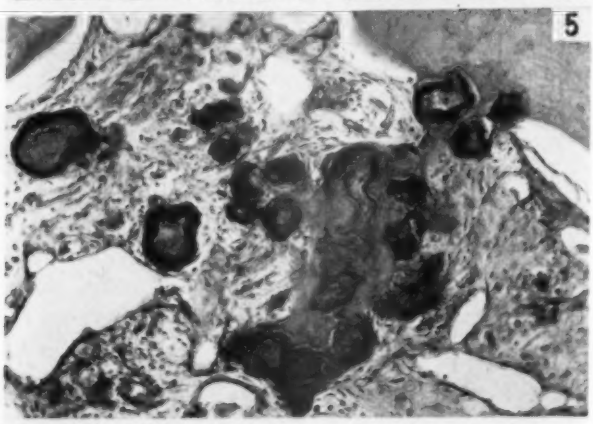
Throughout the marrow, regardless of its nature, the blood vessels are markedly dilated. In addition, distributed throughout, are small hemorrhagic areas with scattered particles of blood pigment. Small zones of myeloid hematopoiesis are occasional findings (Fig. 4). The marrow also contains many osteoclasts, scattered calcific nodules, and fragments of necrotic bone (Fig. 5). Calcification of the media of the arteries and arteriolar sclerosis is noted in the vessels of the marrow (Fig. 6) as well as in those in the connective tissue surrounding the periphery of the bone in one of the cases studied (Fig. 7). (These latter findings are consistent with the autopsy finding of generalized arteriosclerosis in this case.)

B. Relation of Marrow to Bone Formation.—Newly deposited bone is seen along the trabeculae which are surrounded by vascular connective tissue marrow. Bone formation along the trabeculae is diminished in areas where the connective tissue content of the marrow is decreased. Trabeculae, which are surrounded by marrow which is entirely fatty in nature, present no newly deposited bone along their margins.

Definite changes in connective tissue of the marrow accompany the formation of bone. The cells gradually change from a flattened elliptical shape to become oval and, ultimately, attain a polyhedral contour. In this latter stage, they are most commonly seen lining the bone trabeculae and may be considered to be osteoblasts. In the course of bone formation, the finely fibrillar matrix of the connective tissue marrow changes and appears as discrete pinpoint granules (Fig. 8). These granules subsequently fuse and assume the coarse homogeneous appearance of bone matrix (Fig. 9). When bone is laid down along the margin of a trabecula, the alterations in cell morphology, described above, appear to precede the change in matrix. After attaining a polyhedral shape, the cells assume a position along the trabecular margin so that one aspect of the cell is in apposition with the bone and the other is surrounded by marrow. The changes in the marrow matrix follow. With the gradual change in the matrix, that portion of the cell which was formerly surrounded in part by connective tissue becomes progressively embedded in bone matrix until it is completely enclosed by bone (Fig. 10). When surrounded by bone matrix the original connective tissue cell is known as an osteocyte, primarily because of its position rather than because of any pronounced morphologic alteration from the osteoblast stage. With maturation of the bone, the osteocytes become elliptical and flattened.

In addition to the formation of bone along the margins of trabeculae, islands of newly formed bone appear in the connective tissue marrow. Scattered throughout the connective tissue marrow are zones in which islands composed of several elliptical connective tissue cells surrounded by discrete





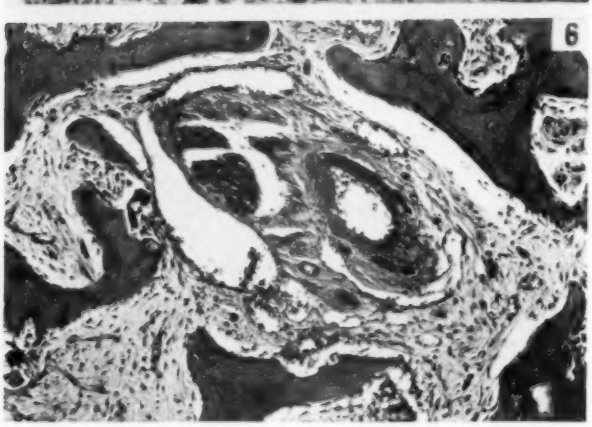
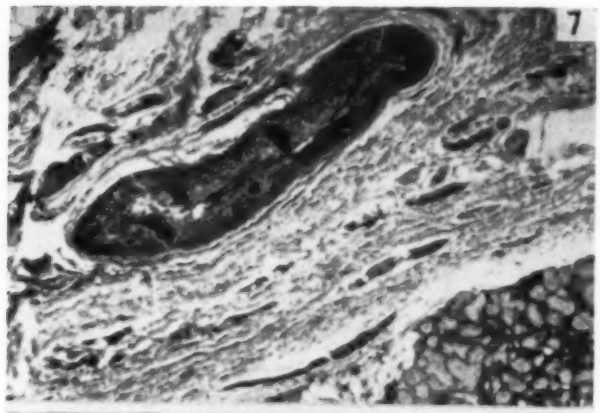
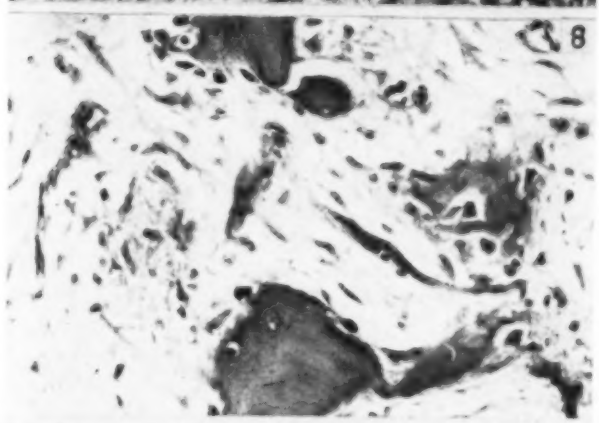


Fig. 4.—Maxilla. Marrow space with dilated vessels, hemorrhage, and a mild degree of hematopoiesis. Amorphous spherical calcified masses are seen on the right. Magnified insert, lower left-hand corner: high-power study of one of the many nucleated red cells in this area. Hematoxylin and eosin. Magnification ×1240.

Fig. 5.—Left maxilla. Calcified fragments of necrotic bone in marrow. Remnant of osteoclast in upper right region apparently calcified in situ along bone margin. Mallory's aniline blue stain for connective tissue. Magnification ×174.

Fig. 6.—Right maxilla. Artery in bone marrow showing zones of calcification in media. Hematoxylin and eosin. Magnification ×150.





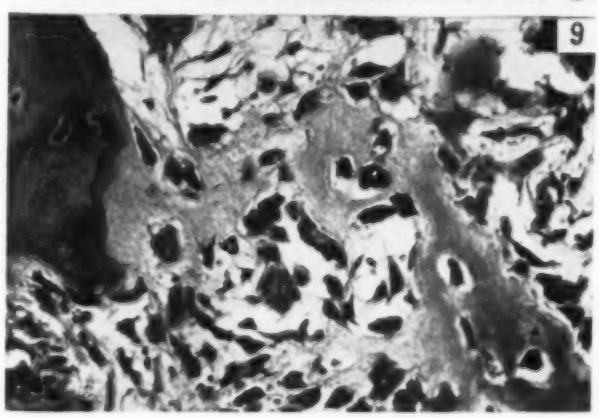


Fig. 7.—Right maxilla. Artery in soft tissue adjacent to gland showing zones of calcification in media. Hematoxylin and eosin. Magnification ×250.

Fig. 8.—Right maxilla. Marrow showing discrete zone comprised of pinpoint granules on the left. Area on right presents three connective tissue marrow cells surrounded by coarse homogeneous matrix. Mallory's aniline blue stain for connective tissue. Magnification ×380.

Fig. 9.—Right maxilla. Pale staining coarse homogeneous matrix forming margin of bone trabecula. Large polyhedral cells enclosed in pale matrix. Hematoxylin and eosin. Magnification ×500.

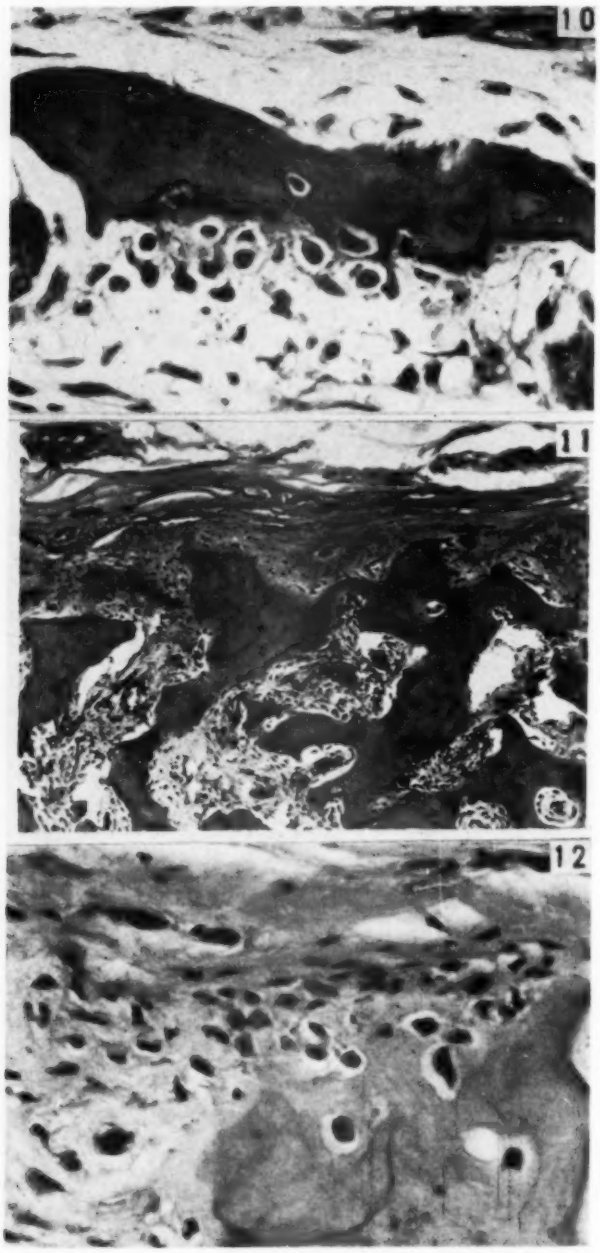


Fig. 10.—Right maxilla. Polyhedral cells adjacent to bone trabecula. Cells in various stages of encapsulation by granular bone matrix. Hematoxylin and eosin. Magnification ×680.

Fig. 11.—Right maxilla. Periosteum and underlying bone trabeculae subjacent to alveolar mucosa. The periosteal cells and fibers blend with the bone matrix. Hematoxylin and eosin. Magnification ×96.

Fig. 12.—Right maxilla. Periosteum and bone. The intercellular connective tissue fibers of the periosteum undergo gradual change and appear as pale staining bone matrix. The elliptical connective tissue cells of the periosteum become polyhedral osteocytes embedded in bone matrix. Hematoxylin and eosin. Magnification ×680.

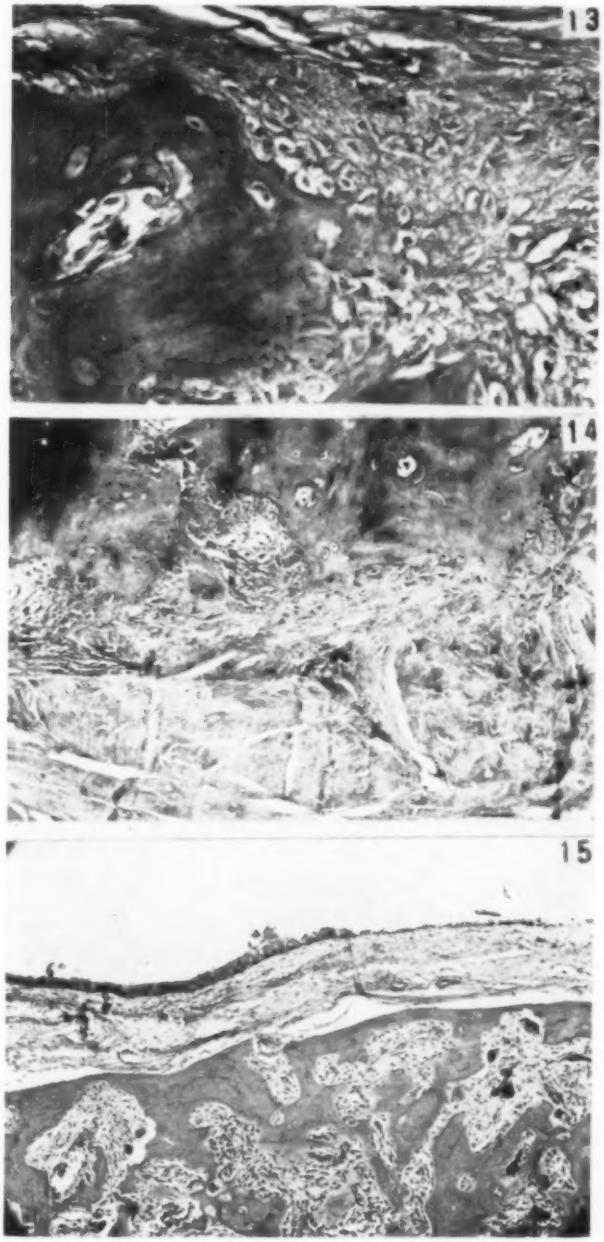


Fig. 13.—Right maxilla. Periosteum and bone. There is a blending of the bone matrix with the intercellular collagen fibers of the periosteum. Mallory's aniline blue stain for connective tissue. Magnification ×300.

Fig. 14.—Upper left maxilla. Tendinous attachment to bone. There is a gradual transition of connective tissue and bone matrix similar in appearance to that shown in Figs. 12 and 13. Hematoxylin and eosin. Magnification ×176.

Fig. 15.—Right maxilla. Lining of sinus and underlying bone. The connective tissue fibers are arranged horizontally in relation to sharply outlined smooth linear bone margin. Hematoxylin and eosin. Magnification ×72.

pin-point granules stand out from the surrounding area (Fig. 8). These granules gradually clump together to form a coarse bone matrix and the elliptical cells become polyhedral in shape and enclosed in individual lacunae which are clearly demarcated in the newly formed matrix (Fig. 9).

C. Periosteum.—In the mandible and maxilla, the connective tissue of the periosteum which surrounds the periphery of the bone cannot be sharply demarcated from the underlying newly formed bone. Both the cells and fibers along the inner aspect of the periosteum undergo gradual changes so that the connective tissue and bone, instead of being clearly outlined from each other, are fused by a series of gradual alterations in cell and intercellular substance. The elliptical cells of the periosteal connective tissue are altered in such a way that the connective tissue cells ultimately become osteocytes (Figs. 11 and 12). Although, from a morphologic standpoint, there is a stage in this transition which conforms with the polyhedral contours characteristic of osteoblasts, this stage merely represents a transitional phase and is in no way separated from the numerous other stages. The intercellular fibers which surround the elliptical connective tissue cells undergo an associated metamorphosis concomitant with the cellular changes and become a moderately coarsely granular bone matrix in which the osteocytes are contained (Fig. 13). This equilibrium and blending between connective tissue and bone is also seen at the attachment of tendons to the bone of the maxilla and mandible (Fig. 14).

The clear-cut demarcation between the subepithelial connective tissue lining the maxillary sinus and underlying bone (Fig. 15) is in sharp contrast with the findings described above in relation to the periosteum surrounding the periphery. The connective tissue cells and fibers of the sinus floor are horizontally disposed and lie in close apposition with a smoothly linear, sharply outlined bone margin.

D. Trabecular Pattern.—The arrangement of the bone trabeculae in Paget's disease can be examined more intelligently when compared with the normal. Normally, in both the mandible (Fig. 16) and maxilla (Fig. 17), the pattern of the bone trabeculae is such that a peripheral cortical layer of bone is demarcated from a central region into which the trabeculae extend in a cancellous pattern. The bone in both the cortical and cancellous regions is lamellated. The lamellae in the cortical region are arranged in lines parallel to the peripheral contours. The lamellae of the cancellous region present no such regular pattern. In both the cortical and cancellous regions, haversian systems containing concentric lamellae may be found.

In Paget's disease the demarcation between the cortical and cancellous regions of the mandible (Fig. 18) and maxilla (Fig. 19) is lost. The bone in both these regions is resorbed and replaced by a regularly arranged, evenly distributed honeycomb of comparatively narrow trabeculae of newly formed bone. Occasional areas are seen in which the cortex has not been completely resorbed and remnants of the regularly lamellated cortical bone remain. In these areas the extent of resorption is marked by an irregular thin line which stains deeply with hematoxylin and describes a semicircular baylike escaloped concavity in the regularly lamellated cortex. Such concavities are often filled with the coarse nonlamellated matrix and polyhedral osteocytes, characteristic of newly formed bone (Fig. 20).

E. Bone Matrix and Osteocytes.—In the trabeculae of newly formed bone, the matrix is coarsely granular in texture and presents no lamellae. The osteocytes are polyhedral in shape with large darkly staining spherical nuclei (Fig. 21). At the periphery of such trabeculae, a border of faintly staining, markedly coarse matrix which blends with the surrounding connective tissue is often observed.

In other areas the bone matrix and osteocytes present a different appearance

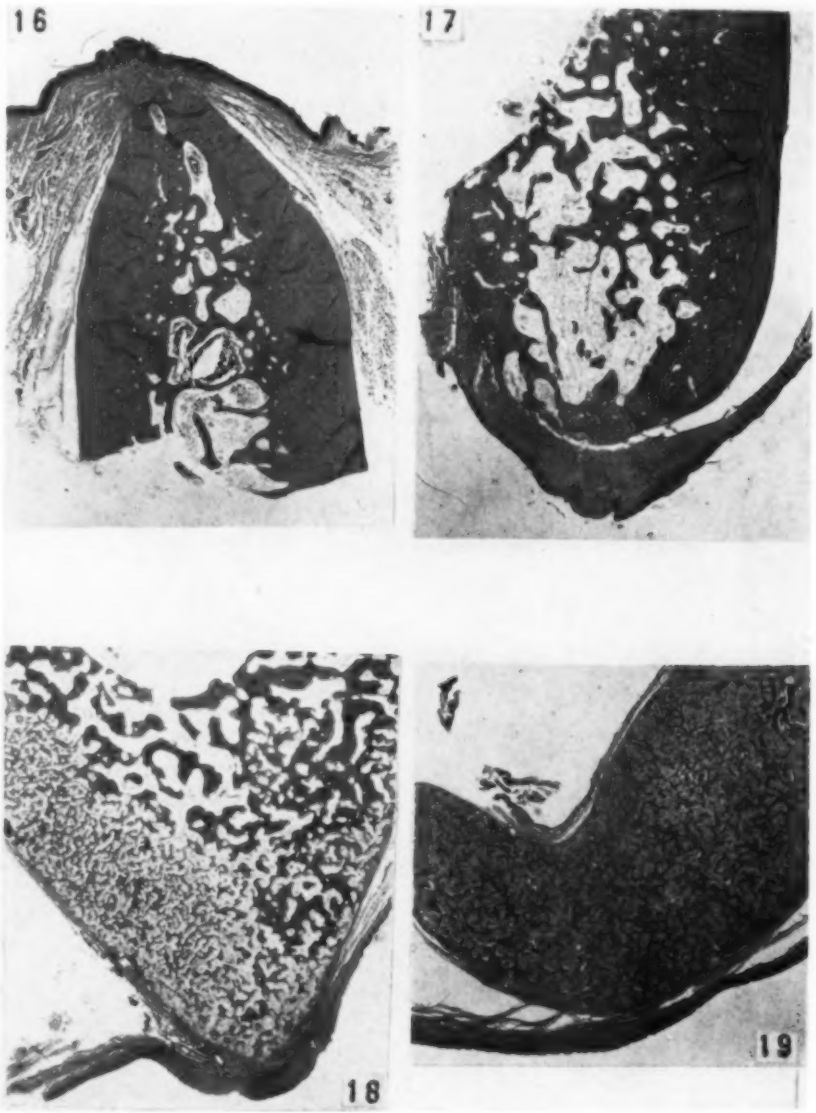


Fig. 16.—Mandible (non-Paget individual). A peripheral cortical layer stands out from the central cancellous region. Hematoxylin and eosin. Magnification ×8.

Fig. 17.—Maxilla (non-Paget individual). A peripheral cortical layer stands out from the central cancellous region. Hematoxylin and eosin. Magnification ×8.

Fig. 18.—Mandible. A demarcation between cortical and cancellous regions is not apparent in Paget's disease. Compare with Fig. 16. Hematoxylin and eosin. Magnification ×8.

Fig. 19.—Right maxilla. The bone trabeculae present a regularly arranged evenly distributed honeycomb pattern. Demarcation between cortical and cancellous regions is absent. Compare with Fig. 17. Hematoxylin and eosin. Magnification ×8.

from that described above in the newly formed bone. The trabeculae are composed of a patchwork of irregularly contoured segments of bone (Fig. 23). The matrix of the segments is generally lamellated and stains clearly with eosin. The osteocytes are elliptical, contain a flattened deeply staining nucleus, and are interspersed in parallel lines conforming with the lamellae. Markedly irregularly curved semicircular cement lines which stain deeply with hematoxylin sharply demarcate the segments of bone from each other. Interspersed between the lamellated segments are areas in which the matrix is coarsely granular, stains with a cloudy bluish tinge, and presents no lamellae. The osteocytes in these latter areas tend to be oval or spherical in shape and are irregularly distributed. The composite mosaic pattern which results from the irregularly arranged segments and curvilinear markings of the saw-toothed cement lines is considered pathognomonic of Paget's disease.

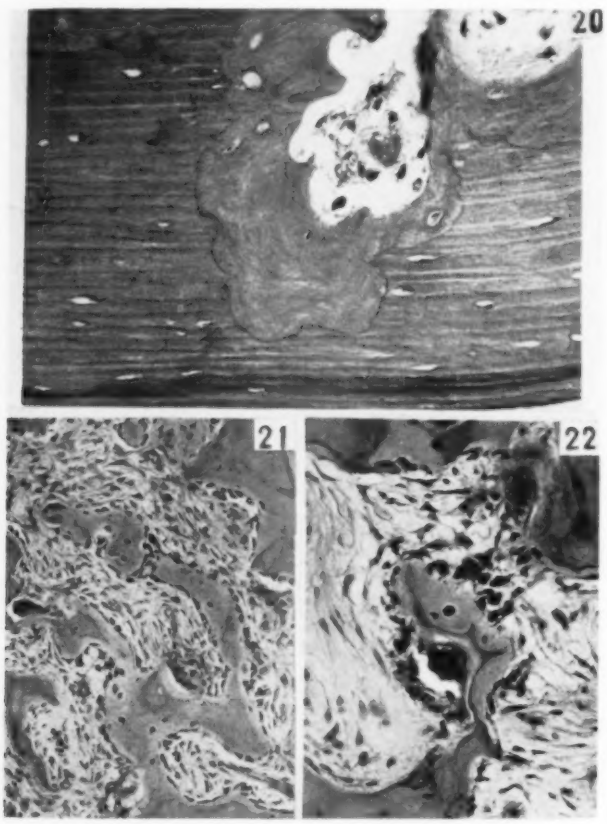


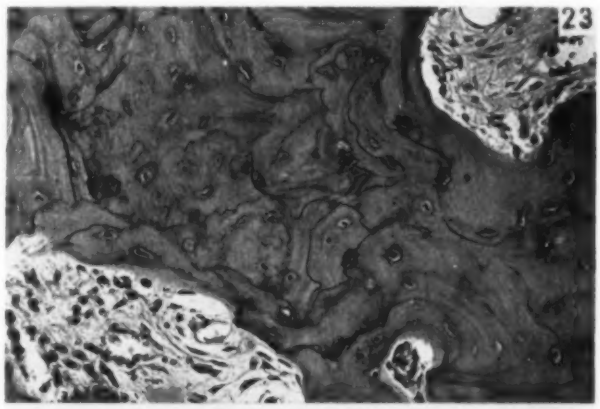
Fig. 20.—Mandible. Remnant of unresorbed regularly lamellated cortical zone containing semicircular baylike escaloped concavity. The concavity is partially filled with coarse nonlamellated bone matrix. Hematoxylin and eosin. Magnification ×380.

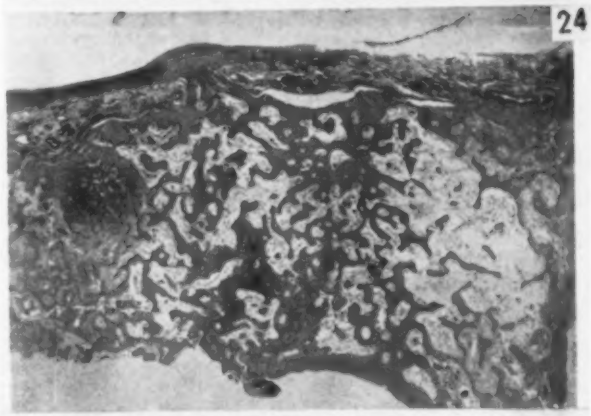
Fig. 21.—Mandible. Bone trabeculae of nonlamellated coarsely granular matrix lined with osteoblasts. The marrow is markedly fibrous. Hematoxylin and eosin. Magnification ×200.

Fig. 22.—Mandible. Thin irregular bone trabecula presenting osteoclast on one aspect and pale staining bone matrix on other. Hematoxylin and eosin. Magnification ×460.

Another feature of the bone matrix is the presence of discrete foci of degeneration (Fig. 24) and increased calcification. The trabeculae in these zones are arranged in small irregular fingerlike projections and stain with hematoxy-

lin and eosin with a hazy bluish tinge which is in marked contrast with the clear pink of the surrounding bone (Fig. 25). The irregularly indented borders of the individual trabeculae in these zones are marked by a continuous thin peripheral line which stains deeply with hematoxylin. Remnants of irregular cement lines are faintly visible in the matrix. The osteocyte lacunae





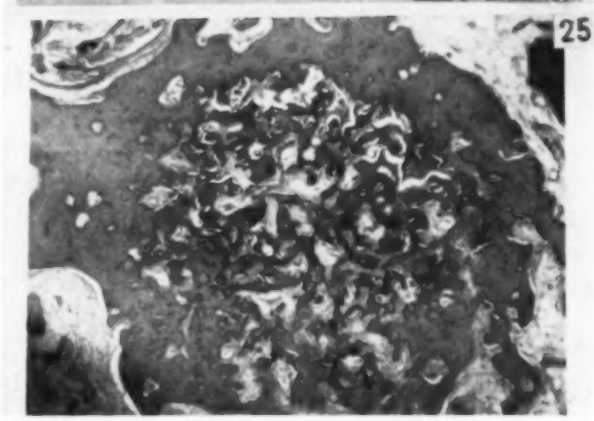


Fig. 23.—Right maxilla. Mosaic pattern of bone characterized by irregularly arranged sawtoothed cement lines. Hematoxylin and eosin. Magnification ×188.

Fig. 24.—Palate. Spherical zone filled with irregular darkly staining fingerlike trabeculae. Hematoxylin and eosin. Magnification ×10.

Fig. 25.—Detail of Fig. 24. Area in bone filled with markedly irregular fingerlike trabeculae which stain more deeply with hematoxylin than bone in surrounding area. Hematoxylin and eosin. Magnification ×24.

are enlarged and present minutely irregular margins. Osteocytes are absent. Under low-power examination, the zones of degeneration stand out sharply from the surrounding bone. High-power study, however, reveals no clear-cut outline of demarcation. The surrounding bone is progressively altered in the direction of the center of the degenerate area in such a way that the matrix gradually loses its clear pink appearance and assumes a bluish tinge, the osteocyte lacunae become enlarged, the osteocytes become shriveled and distorted as the bone undergoes degeneration and assumes the appearance described above.

F. Osteoclasts.—Osteoclasts are extremely common in the zones of newly formed bone in Paget's disease. They are found in the marrow, in covelike depressions in close apposition to trabecular margins, and encompassing the tips of bone trabeculae (Fig. 26). They are comparatively rare in areas in which bone formation is not taking place. They are spherical in shape. The cytoplasm is coarsely granular and contains an occasional vacuole and a variable number of round darkly-staining nuclei which are distributed throughout the cell in no regular pattern.

The osteoclasts present a varied picture. In some instances where they lie free in the connective tissue marrow, they are shrunken and surrounded by concentric clear zones which demarcate them from an encapsulating layer of connective tissue cells. The cytoplasm stains faintly and is fragmented, the nuclei appear hyperchromic and pyknotic. In other areas, the clear zone around the osteoclasts is enlarged. The peripheral portion of the cytoplasm is markedly filamented and is scattered throughout the clear zone. A markedly vacuolated ring of cytoplasm is often seen separating the filamented cytoplasmic debris from the central portion of the cytoplasm (Fig. 27). In some instances there is no remnant of unaltered cytoplasm (Fig. 28). The entire cytoplasmic mass may be markedly vacuolated, or filamented, and present varying degrees of fragmentation. In addition to these cytoplasmic changes, the nuclei may be shrunken and hyperchromic.

When the osteoclasts are in apposition with a bone surface on one aspect and connective tissue marrow on the other, the clear zone described above does not encompass the entire cell but appears as a half moon separating the osteoclasts from the encapsulating layer of connective tissue cells (Fig. 29). In these instances the peripheral cytoplasm which is adjacent to the connective tissue cells is finely granular and fragmented. The osteoclasts can be divided into a peripheral portion which is adjacent to the connective tissue margin, and an intermediate region which separates the peripheral portion from the nuclei-containing zone which lies in a covelike depression in the bone. The peripheral portion is finely granular and in part fragmented, the intermediate portion is markedly vacuolated, and the nuclear portion is not visibly altered. The nuclei vary in appearance from normal to a hyperchromic pyknosis.

Associated with these alterations in the osteoclasts are changes in the encapsulating ring of connective tissue cells noted above. Originally thinned out and flattened, these cells attain the polyhedral appearance characteristic of osteoblasts. These osteoblasts often become embedded in newly deposited

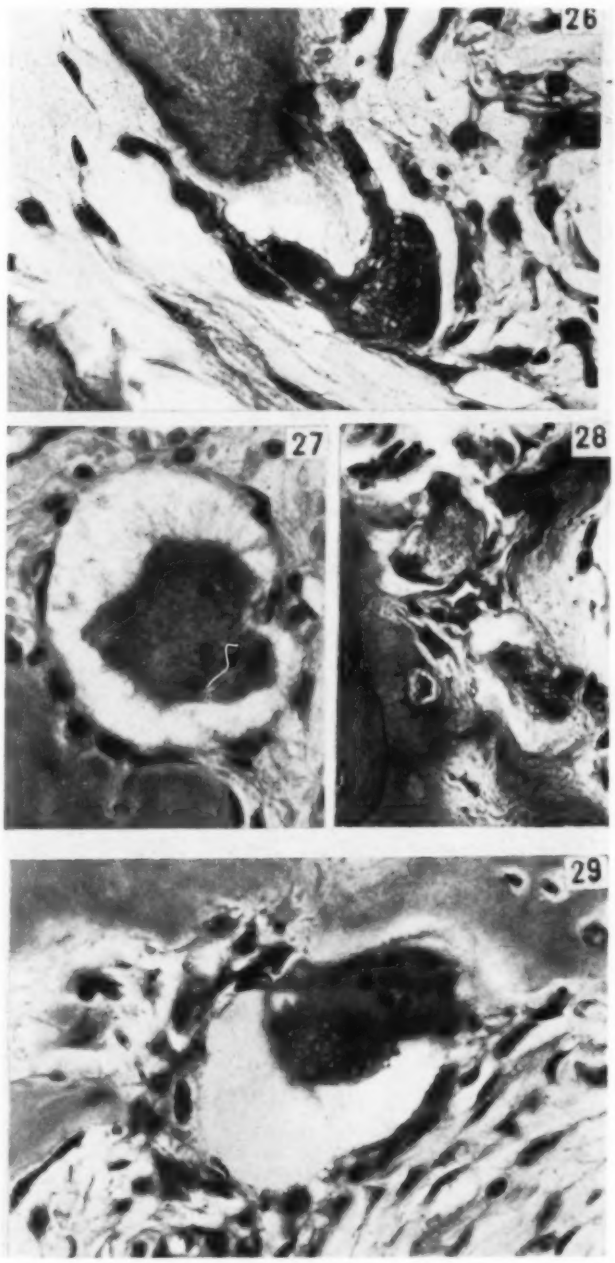


Fig. 26.—Right maxilla. Horseshoe-shaped osteoclast conforming to contour of bone trabecula. Pale staining fragmented amorphous debris continuous with bone matrix. Mallory's aniline blue stain for connective tissue. Magnification ×880.

Fig. 27.—Mandible. Osteoclast showing vacuolated and filamentous cytoplasmic debris separating central mass of cytoplasm from peripheral layer of surrounding connective tissue cells. Hematoxylin and eosin. Magnification ×680.

Fig. 28.—Mandible. Remnants of osteoclasts. The nuclei are barely discernible in the fragmented vacuolated cytoplasmic debris. Mallory's aniline blue stain for connective tissue. Magnification ×460.

Fig. 29.—Left maxilla. Osteoclast adjacent to bone surface. The cytoplasm in relation to the surrounding ring of connective tissue cells is finely granular and fragmented and is separated from the nuclear containing cytoplasm by an intermediate vacuolated zone. Hematoxylin and eosin. Magnification ×680.

bone matrix so that the osteoclast ultimately becomes encapsulated by a zone of newly formed bone (Figs. 30 and 31).

The composite alteration in osteoclasts described above is that of shrinkage of the cell, the appearance of an encircling clear halo which separates the cell from an encapsulating layer of connective tissue cells, peripheral vacuolar degeneration of the cytoplasm, subsequent fragmentation and dissolution of the cytoplasm with the progressive enlargement of the encircling clear zone, progressive central extension of degeneration with ultimate involvement of the entire cytoplasm, and pyknosis of the nuclei. Concomitant with these changes in the osteoclast, the encapsulating connective tissue cells undergo a cytomorphosis to the osteoblast stage and become embedded in newly formed bone, simultaneously enclosing the degenerating osteoclast. Occasionally, fragmented remnants of osteoclasts are observed in the marrow spaces, while in other instances they appear to have been calcified in situ along a bone margin (Fig. 5).

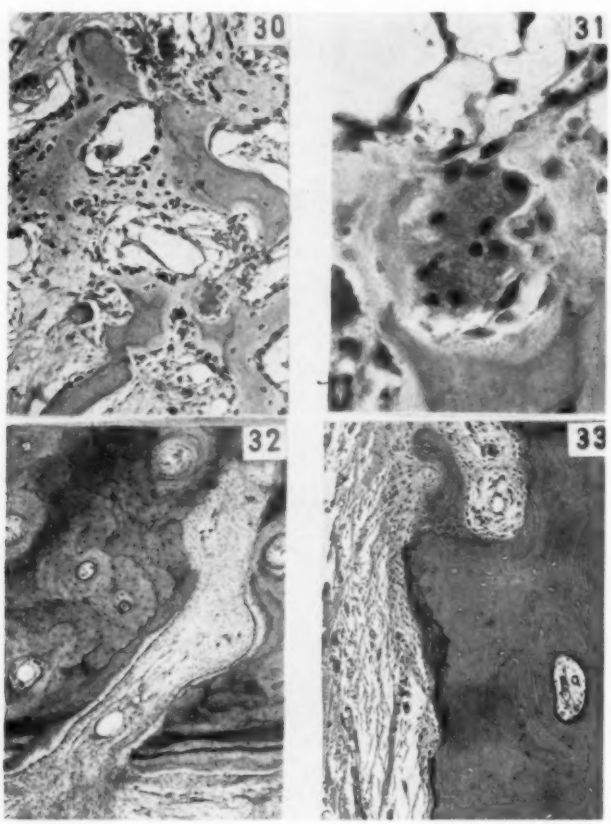


Fig. 30.—Mandible. Cells surrounding osteoclast embedded in pale staining bone matrix. In addition, note gradual transition as connective tissue on left blends with pale staining bone matrix. Hematoxylin and eosin. Magnification ×100.

Fig. 31.—Detail of Fig. 30. Osteoclast surrounded by pale staining bone matrix containing embedded connective tissue cells. Hematoxylin and eosin. Magnification ×520.

Fig. 32.—Mandible (non-Paget individual). Bone margin lined with pale staining matrix and osteoblasts in one area and irregularly escaloped covelike concavities in another. Hematoxylin and eosin. Magnification ×150.

Fig. 33.—Mandible (non-Paget individual). In one region the bone blends with the connective tissue of the periosteum. In another the bone is sharply demarcated from the periosteum by an irregular margin. Hematoxylin and eosin. Magnification ×100.

DISCUSSION

A detailed description of the microscopic findings in the maxilla, mandible, and palate in Paget's disease is presented above. No bone changes are described which are peculiar to these bones which have not been noted in other parts of the skeletal system in Paget's disease. This fact demonstrates the basic similarity between the bones of the jaws and bones elsewhere in the body.

The contrast in the bone picture in various closely associated regions of the same bone is an outstanding finding. There is no apparent rationale for the presence of bone formation, bone resorption, degeneration, and inactivity either blending with each other or appearing as discrete isolated foci. Areas comprised of a regular network of trabeculae of nonlamellated bone replace previously existing bone which has undergone resorption in both the cancellous and cortical regions. The marrow surrounding the newly formed trabeculae is comprised of vascular connective tissue, the cellular and intercellular components of which undergo alterations accompanying bone formation. The fact that there is no bone formation in those areas in which the marrow is composed of fat cells, in contrast to the pronounced formation associated with the connective tissue marrow, points to a relationship between the connective tissue marrow and bone formation. The tendency on the part of the connective tissue to become morphologically altered to facilitate bone formation is an important feature of Paget's disease in the jaws.

Bone formation in relation to cells arising from connective tissue contained within the bone is a feature of Paget's disease which is also observed in the jaws of non-Paget individuals. It is the marked predominence and generalized distribution of such bone formation which characterizes Paget's disease.

Periosteal bone formation, and a blending of the periosteal connective tissue with the underlying bone, have been described above subjacent to the oral mucosa and absent in relation to the connective tissue underlying the maxillary sinus. It may, therefore, be inferred that function exerts a stimulating effect upon the bone formative processes in Paget's disease. Similar findings have been demonstrated in relation to periostea in jaws of non-Paget individuals[5] (Fig. 33). In Paget's disease, however, the periosteal formation subjacent to the oral mucosa is generalized, whereas it occurs only in isolated areas in non-Paget individuals. In addition, the extent of such bone formation is noticeably more pronounced in Paget's disease.

The microscopic appearance of bone matrix indicates to a certain degree the nature of the bone activity in a given area. As previously noted, the bone matrix varies throughout the jaws in Paget's disease, representing concomitantly bone formation, bone resorption, and bone degeneration heterogenously distributed in close proximity to each other. Isolated areas of pronounced bone formation and resorption are bordered by zones of inactivity. Inactive zones showing signs of previous activity may become the sites of marked bone formation and resorption. Paget's disease, therefore, represents not a series of progressive alterations but rather the sporadic recurrence of activity and quiescence. The variable bone picture in Paget's disease is possible because of the extremely flexible nature of bone morphology. The presence of bone

formation and bone resorption in close association is also a common finding in the jaws of non-Paget individuals (Fig. 32). The mosaic bone pattern considered diagnostic of Paget's disease may be interpreted as a graphic record of repeatedly interrupted alternating periods of bone resorption and formation which have previously occurred in a given area. Alternating periods of resorption and formation (Fig. 22) occur in a single area with such frequency as to prohibit the formation of trabeculae composed entirely of regularly lamellated bone. The pattern of the bone matrix in Paget's disease differs from that observed in non-Paget individuals, not in the nature of the bone matrix and cells, but in the distribution and size of the segments of otherwise normally appearing bone.

The presence of covelike indentations along the periphery of bone trabeculae is the only morphologic evidence of bone resorption described above. Although many of these indentations are filled with connective tissue cells which may be related to the resorption process, the greatest number of them contain multinuclear osteoclasts. Alterations in the matrix of bone partially enclosed by an osteoclast have been shown here (Fig. 26). It may be assumed that the osteoclast is responsible in some degree for these alterations in bone matrix. Osteoclasts are found in greatest numbers in the zones of active bone formation and are infrequent where there is no osteogenic activity. This suggests an attempt to balance the bone equilibrium by increasing resorption in order to compensate for the predominant formative activity. Although osteoclasts are seen in the jaws in non-Paget individuals, they occur in far greater numbers in Paget's disease.

The alterations described in the cytoplasm and nuclei of the osteoclasts and the associated changes in the connective tissue cells of the marrow which encapsulate them are of interest. Similar changes, less pronounced in nature and distribution, are seen in the osteoclasts in non-Paget individuals. It appears feasible to interpret these changes in the osteoclasts as degenerative in nature. The changes in the connective tissue cells which surround the osteoclasts as they undergo degeneration, parallel those seen in the cells of the connective tissue marrow in other areas. As previously noted, preceding or associated with bone formation, the connective tissue cells of the marrow change from a thin flattened elongated shape to a polyhedral form. The finding of these changes in the connective tissue cells which surround the osteoclasts, plus the subsequent deposition of the bone in relation to the connective tissue cells with apparent encapsulation of the osteoclasts (Figs. 30 and 31), is further evidence of the preponderant tendency toward bone formation which exists in areas of the jaw in Paget's disease.

SUMMARY

Microscopic study of the maxilla, mandible, and palate in Paget's disease revealed the following features:

Bone formation, resorption, degeneration, and inactivity heterogeneously distributed in close proximity to each other.

Alteration of the previously existing bone pattern and its replacement by a honeycomb of newly formed bone trabeculae.

Replacement of fatty marrow by vascular connective tissue in accordance with no regular pattern.

Pronounced bone formation associated with connective tissue marrow and absence of formation, associated with fatty marrow.

Pronounced osteoclastic activity, and degeneration in zones of bone formation.

Scattered areas in which the bone presents a mosaic pattern as the result of recurrent opposition and resorption.

Isolated foci of bone degeneration and increased calcification.

The changes observed in the maxilla, mandible, and palate were similar to those described in the remainder of the skeletal system in Paget's disease.

A comparison of the findings in Paget's disease with those observed in the mandible and maxilla in non-Paget individuals of a similar age group revealed that all of the individual histologic changes observed in Paget's disease could be duplicated in non-Paget individuals. Although Paget's disease is recognizable as a composite histologic entity, it represents an alteration in severity and pattern, but not in the basic nature of the detailed histologic changes which generally occur in the mandible and maxilla in non-Paget individuals.

The author is indebted to Lt. Com. H. Spencer Glidden, U.S.N.R., instructor in Pathology and Bacteriology, Tufts College Medical and Dental School for making one of the cases available for study, and to Anna Morse, M.S., instructor in Oral Pathology, Tufts College Dental School for the preparation of tissue sections.

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INFECTED GLOBULOMAXILLARY CYST

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E. P., a white man, 40 years of age, was advised in November, 1940, to have a cyst of the anterior maxillary region removed. He declined treatment and did not return to Washington University Dental Clinic until March, 1941.

Physical Examination.—The patient had a swelling of the tissues overlying the roots of the right anterior maxillary teeth. A small opening, surrounded by an elevated, red area was observed between the right maxillary second incisor and the right canine. Pressure on the adjacent region gave a sensation of being fluctuant and a few drops of creamy, yellow fluid exuded from the fistulous opening. There was no pain on pressure and only slight tenderness. The right maxillary first incisor and canine responded readily to application of ice but the second incisor gave no response. The first premolar in this region had been subjected to root canal therapy. All maxillary teeth present in the mouth were affected by periodontitis and many of the mandibular teeth were similarly diseased.

Radiographic Examination.—Intraoral films show a radiolucent area extending from the second incisor to the area of the first premolar (which had been extracted some time before November, 1940). This area is not spherical but is irregularly hemispherical (Fig. 1). It measures 1½ by 2½ cm. in the canine view. The roots of the second incisor and canine are separated more than ½ cm. at the apex although these teeth contact each other in the coronal portion. The canine appears to lie against the first premolar. The first premolar shows partial root canal filling.

Operation.—Local anesthesia was used (March 4, 1941), and when the lingual injection was made, pus appeared on the labial of the right anterior maxillary teeth. An incision was made and the mucosa dissected from the labial surface of the bone overlying the incisors and canine. A membrane was visible through a defect in the bone between the canine and second incisor. This opening was enlarged and the cystic membrane was freed from the surrounding tissues by blunt dissection. It extended to the palatal bone posteriorly, the nasal floor superiorly, the alveolar mucosa anteriorly, and to the alveolar crest inferiorly. It was necessary to free the fistula on the anterior surface by incision. When the membrane was dissected at its nasal border, a small amount of blood escaped from the nostril. The first incisor, second incisor, canine, and first premolar were removed, and the tissue sutured to place with an iodoform gauze drain inserted into the defect.

Two days later the patient reported that he was now able to breathe freely through the right nostril for the first time in about two years, although he

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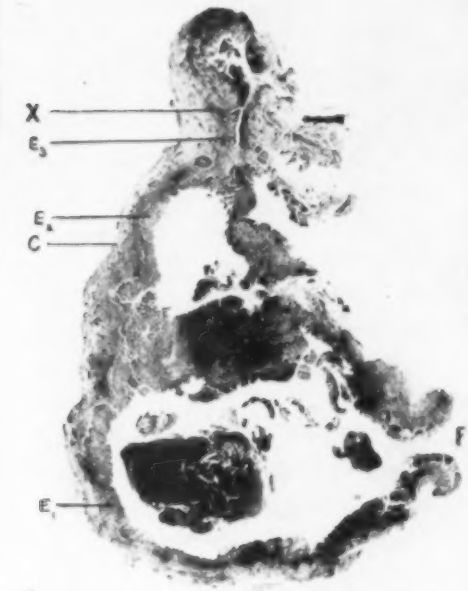

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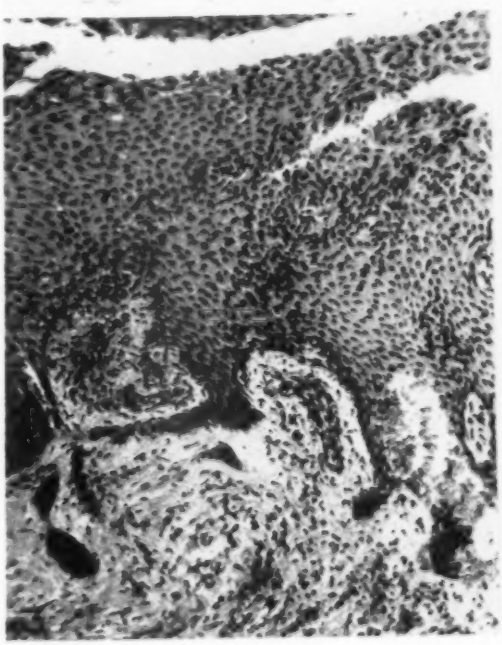

Fig. 1.—Intraoral radiograph of right canine area. Note separation of canine and second incisor roots; partial root canal filling in first premolar.

Fig. 2.—Photomicrograph of section through lesion in Fig. 1. *C*, Connective tissue capsule; E_1, E_2, E_3, squamous epithelium lining cyst; *F*, fistula; *X*, area of ciliated, columnar epithelium seen in Fig. 4.

Fig. 3.—Stratified squamous epithelium from lining of cyst adjacent to area *X* in Fig. 2.

Fig. 4.—Ciliated columnar epithelium from area *X* in Fig. 2.

had not complained to us of any breathing difficulty prior to the operation. There was little swelling, and irrigation and drainage were continued for two weeks. The patient returned for extraction of other teeth and construction of dentures. On May 5, 1942, when last seen, he showed no evidence of recurrence and the operative defect was barely observable.

Pathologic Examination.—The specimen, as examined microscopically, consisted (Fig. 2) of a connective tissue capsule (*C*), enclosing three cavities. The inferior cavity was largest and shows a squamous epithelial lined opening (*F*). This large cavity was lined by stratified squamous epithelium (E_1) which projected into the surrounding connective tissue and extended superiorly to join with the epithelium lining the second cavity. There was debris consisting of laked erythrocytes and disintegrating leucocytes present within the cavity. The cyst superior to this was lined by similar stratified epithelium (E_2) which was continuous with that of the inferior cyst. The tissue between these two cavities contained heavy fibrous strands, polymorphonuclear leucocytes, lymphocytes, and dilated blood vessels. The superior cavity was lined with stratified squamous epithelium (Fig. 2 E_3 and Fig. 3) but had two diverticulae (Figs. 2 and 4) which were lined with ciliated columnar epithelium (Fig. 4). The columnar epithelium blended into the squamous epithelium without any sharp line of demarcation. The diagnosis was inflamed epithelial-lined cyst, compatible with the clinical diagnosis of dentoperiosteal cyst.

COMMENT

This cyst was considered of dentoperiosteal type (radicular cyst), arising from the pulpless first premolar or from the pulpless second incisor. A photomicrograph of the cyst and a reproduction of an intraoral radiograph were included in a report on the diagnosis of cysts of the jaws.* In that paper this lesion is described as a dentoperiosteal (radicular) cyst. In examining enlarged photomicrographs and radiographs of the lesion, Dr. K. H. Thoma suggested the possibility of globulomaxillary cyst (June 7, 1943). The case has been reviewed since then and the radiographs and sections carefully studied. We believe the diagnosis of this fissural type cyst is compatible with the clinical, radiographic, and microscopic evidence.

Globulomaxillary (fissural, nasoalveolar) cysts are differentiated from dentoperiosteal (radicular, dental root) cysts chiefly by radiographic evidence. The fissural cyst is formed between the second incisor and canine and spreads the roots of these teeth by its growth, while the dental root cyst forms in the periapex and does not tend to separate the teeth. The teeth in the area of a globulomaxillary cyst are not necessarily included in the defect and the lamina dura may be intact on all teeth, but the radicular cyst must include at least one tooth root which shows a break in its lamina dura. Differentiation from the parodontal type of dentoperiosteal cyst is not as simple but a history of parodontal infection or abscess may serve as a guide. Dentigerous cysts are readily differentiated because they include the crown of a tooth or an anomalous tooth (odontoma) and follicular cysts are rare in this region and position.

*Robinson, H. B. G., and Koch, W. E., Jr.: Diagnosis of Cysts of the Jaws, J. Missouri State Dent. Ass. 21: 187, 1941.

The ameloblastoma (adamantinoma, adamantoblastoma) is different radiographically because it produces a lobulated area or a scallop-bordered area and its microscopic appearance is quite characteristic.

That this lesion was diagnosed as a dentoperiosteal cyst is readily understood because two teeth within the area of bone destruction were pulpless. The separation of the roots was overlooked. It was hypothesized that the cyst expanded to the nasal floor where it was invaded by ciliated, columnar epithelium. If the diagnosis of globulomaxillary cyst is accepted, this strained theory is unnecessary. The globulomaxillary cyst is formed from remnants of the fetal epithelium which occupied the fissure between the globular process (which forms the premaxilla) and the maxillary process (which forms the rest of the maxilla). This fissure is closed in prenatal life, or a cleft jaw results. The fissural epithelium is partly oral (stratified squamous) and partly nasal (ciliated, columnar). We believe this lesion was an infected globulomaxillary cyst.

This case serves to illustrate how easily the diagnosis of globulomaxillary cyst may be overlooked. Differential diagnosis may be important, clinically, because extraction of teeth in an area of a globulomaxillary cyst is not indicated, per se. In this case the teeth were removed because of periodontal disease.

SUMMARY

A cyst, occurring in the anterior region of the maxilla, is reported. Although it contained both stratified, squamous, and ciliated columnar epithelium, and separated the second incisor and canine roots, a diagnosis of dentoperiosteal cyst was made because of pulpless teeth in the area. The clinical, radiographic, and microscopic evidences are more compatible with a diagnosis of infected globulomaxillary cyst.

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THE DENTIST'S RESPONSIBILITY IN ORAL TUMORS

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ORAL malignancies account for approximately 30 per cent of deaths caused by cancer.[1] However, malignant tumors of the mouth present a reasonably good prognosis if properly treated in an early stage. The discrepancy in these statements must be attributed to a failure in recognizing early pathologic changes of the mucous membranes in the oral cavity. The dentist has a great opportunity for repeated examination of his patient, and the important role he plays in the management and early diagnosis of oral malignancies is readily apparent. If the dentist recognizes pathologic lesions of the mouth and properly advises therapy, his contribution to the control of oral malignancies will be invaluable.

The dental profession has long been criticized for restricting examination of patients to the finding of cavities in the teeth and roots in edentulous areas; but dentistry has now developed to a point where the mouth and its structures are its field and responsibility, and warrant consideration as such. No phase of dentistry offers more opportunity to be of real service than that of being on the alert for, detecting, and properly handling tumors of the mouth. This places a large responsibility on the profession, for it is the dentist, rather than the physician, who sees patients with oral malignancies while in a curable stage. Procrastination or ignorance on his part spells doom for the patient.

An examination of the mouth should extend from the vermilion border of the lips to the oropharynx, with particular attention given to the soft tissues and supporting mucosa of the alveolar ridge. This does not mean an invasion of other fields or an attempt to diagnose general disturbances associated with the teeth. It simply means that the dentist of today, as an oral diagnostician, should be capable of serving the patient more adequately, and as a consequence a closer relationship between dentist and physician will result.

Martin,[2] in his recent series of patients with oral tumors, found that 25 per cent first consulted a dentist; 15 per cent had had teeth extracted three months prior to his observation, and 10 per cent attributed the growth to ill-fitting dentures. Singer[3] says that 4 per cent of the malignant tumors of the body originate in the mouth. Gernez[4] states that the majority of all patients with cancer of the gums first consult a dentist; and practically all of Kirkham's[5] cases had been erroneously diagnosed as dental abscesses before admission to the hospital. Epitheliomas of the maxillary sinuses develop in close contact with the dental roots and their nerve supply, and the patient may seek consultation because of pain of dental origin. In such cases extraction of the elongated teeth offers no relief, and the pain persists. del Regato[6] gives a complete and comprehensive study of the sites of origin of these tumors and their relation to the dental structures.

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The biopsy is a valuable adjunct in the diagnosis of any mouth lesion and is not used nearly as extensively by the dental profession as it should be. There is much discussion concerning the danger of spreading of the tumor when a biopsy is made of malignant lesions. As yet, we have no proof to indicate such danger exists. All lesions of the oral cavity, regardless of their appearance, should be biopsied and studied by a competent pathologist.

The technique of the biopsy is a simple procedure, but the site from which the material is taken is important and necessitates consideration. Material from the center of the lesion is frequently only amorphous debris with no definite histologic picture. For this reason, a portion of normal tissue adjacent to the lesion should always be included for study.

For most biopsies of the mouth, only a slight amount of anesthesia is necessary; 2 per cent novocain, as used in extraction, is adequate. Novocain should not be injected directly into a lesion. The site should be cleansed with an alcohol sponge and the novocain injected into the normal tissue at the edge of the area to be excised. The incision should be elliptical, starting from the center of the lesion and extending out into normal mucosa. The important point to remember is to extend the incision deep into the subcutaneous tissues. The pathologist's report will be based only on the tissue which he receives.

The following cases are presented to illustrate the need for education of the public to an appreciation of the malignant possibilities of various oral lesions and the necessity of early consultation; also, they indicate the need for increased diagnostic acumen on the part of the physician and dentist, and the institution of earlier and more effective therapeutic measures.

Fig. 1.—Case 1. Precancerous lesion of the lower lip.

Case 1.—G. W., a 58-year-old white male, referred by a physician, was first seen in the clinic in May, 1942. There was a lesion on the lower lip, 1.5 by 1.5 cm., which had been present for two years (Fig. 1). The lip had bled easily and healed intermittently. Our first pathologic report was "hyperkeratinization, hyperplasia, and chronic inflammation. Suggest another biopsy." A second biopsy showed breaking through of basement membrane. The suggestion was made that it should be treated as cancer. The patient received 3,000 r. units of radiotherapy. In November, 1942, the lip was normal with no nodes palpable in the neck.

Comment.—This case represents a precancerous lesion which was recognized and treated before any malignant changes were present. The patient will be re-examined periodically, but the lesion is considered cured.

Case 2.—M. S., an 80-year-old white male, referred by a physician, was first seen in the clinic in February, 1942. There was a large growth on the lower lip measuring 3 by 4 cm. which had been present for two years. When the lesion reached its present size, there was discomfort in eating. Examination revealed a large, strawberry-colored lesion on the lower lip (Fig. 2). The teeth were in very poor repair, and no neck nodes were palpable. Biopsy report was epidermoid carcinoma, Grade I. All remaining teeth were extracted, after which 3,000 r units of x-ray irradiation were given. The patient was last seen one year after treatment, with no evidence of any recurrence.

Comment.—In sharp contrast to Case 1, this lesion had been present for two years and had undergone malignant changes, which responded to x-ray therapy. Because it was a late lesion of the lip, it must be watched closely for evidence of local recurrence and lymph node metastasis.

Fig. 2.—Case 2. Epidermoid carcinoma.

Case 3.—W. R., a 64-year-old white male, referred by a physician, was first seen in the clinic in April, 1942. The chief complaint was a small lump under the tongue which had been present for two and a half years with a slow increase in size. Examination revealed a superficially ulcerated and indurated area measuring about 2 cm. in its long diameter located in the anterior floor of the mouth to the right of the frenum (Fig. 3). The teeth were carious and in poor repair. In the right submaxillary space, several small soft nodes were palpable, but they did not feel as if they were metastatic. Oblique x-ray views of both mandibles were noncontributory. Biopsy showed epidermoid carcinoma, Grade II. All the remaining teeth were extracted, and the patient then received a total of 2,572 r units of x-ray therapy. In August, 1942, the patient was readmitted for an exploration of a suspicious node in the left submaxillary triangle. Microscopic examination showed only chronic inflammation of the submaxillary salivary gland. Four months prior to his visit in April, 1943, the patient stated that his local dentist had told him he had a retained root tip in his right mandible and had operated to remove it. An examination revealed an area

of exposed alveolar bone with evidence of necrosis (Fig. 4). X-ray report at this time showed destructive changes in the alveolar ridge and anterior portion of the right mandible. He was advised of the home care of the mouth and told to return for re-examination in three months.

Comment.—This early lesion of the floor of the mouth responded satisfactorily to x-ray therapy and the post-treatment course would have been uneventful if there had been no surgical interference by his dentist. This case well illustrates the tragic consequences of the extraction of teeth or any surgical interference in bone that has been heavily irradiated. The dangers involved in any operative procedure upon avascularized bone cannot be stressed too emphatically, and it must be remembered that the longer the period following irradiation, the more avascular the bone becomes. *There is no safe period for surgical procedures upon irradiated jaw bone.*

Fig. 3.—Case 3. Early lesion in right anterior floor of the mouth.

Fig. 4.—Case 3. Radionecrosis of the irradiated mandible following unnecessary surgical interference.

Case 4.—S. B., a 43-year-old white male, referred by a physician, was first seen in the clinic in September, 1941. The chief complaint was a large, indurated, ulcerating lesion in the floor of the mouth. Twelve years ago, a wart-like growth had appeared on the right posterior two-thirds of the tongue. In the last few months previous to our examination, the lesion grew rapidly and bled easily and was painful. Examination of the mouth showed the teeth to be

very dirty and in poor repair. On the right posterior two-thirds of the tongue was a bleeding, indurated, fungating mass extending along the anterior inferior border. The floor of the mouth was indurated and the tongue fixed (Fig. 5). Bilateral adenopathy was palpable in the neck. The biopsy of the lesion showed epidermoid carcinoma, ungraded. The patient refused to have his teeth extracted. He received 2,500 r units of x-ray irradiation over two fields. He was last examined in August, 1942, at which time there was no evidence of tumor. The patient has since expired with reported extensive necrosis of the mandible.

Comment.—This case illustrates a far-advanced lesion on the floor of the mouth. The rapid regression of the lesion with therapy was surprising, and the end result would have been more favorable had the patient permitted the extraction of his teeth previous to radiation therapy. The irradiation undoubtedly contributed to the extensive necrosis of the mandible.

Fig. 5.—Case 4. Late lesion in the floor of the mouth.

Case 5.—H. W., a 78-year-old white male, referred by a physician, was first seen in the clinic in May, 1941. Eight months before admission to the hospital, a sore spot had developed on the left side of the tongue which two months later became hard, tender, and ulcerated. Examination revealed an area 3 cm. in diameter on the left half of the central area of the tongue but not involving the floor of the mouth. It was indurated and superficially ulcerated (Fig. 6). The mouth was edentulous, and there were no palpable lymph nodes in the neck. The biopsy report was epidermoid carcinoma, Grade I. A total of 184 mg. hours of radium irradiation was given. In August, 1941, the tongue showed pale scarring and telangiectasis along the left border, which suggested tumor. On the left side of the tongue was a hard mass of tissue measuring 1.5 by 1 cm., the center of which was depressed and covered with slough, while the edges were raised and granular. Biopsy showed epidermoid carcinoma, ungraded, and radiation reaction. In September, 1941, the patient had a hemiglossectomy. In April, 1943, the tongue was pliable with no evidence of disease, no pain, and no evidence of cervical adenopathy (Fig. 7).

Comment.—This was a relatively early lesion of the tongue which did not respond to interstitial radiation and was subsequently excised. It must be remembered that malignant lesions of the tongue and floor of the mouth are the most formidable of those that occur within the oral cavity, and are prone to

metastasize more promptly. The lateral borders of the tongue are most frequently involved, probably because they are subjected to the greatest trauma. Growths on the border of the tongue offer a better prognosis than those on the ventral portion of the tongue and the floor of the mouth because of the rapid local extension and metastasis of the latter.

Fig. 6.—Case 5. Cancer of the tongue.

Fig. 7.—Case 5. Photograph two years following hemiglossectomy.

Case 6.—F. J., a 66-year-old white male, referred by his physician, first attended the clinic in March, 1941. There were white patches at the base of the tongue which were first noticed five weeks before. Examination of the mouth revealed a soft, white area, 1.5 cm. wide at the base of the tongue, which extended bilaterally in the floor of the mouth to the first molar region (Fig. 8). The remaining teeth were quite carious. The patient had been a confirmed user of chewing tobacco for fifty years. The biopsy showed leucoplakia of the floor of the mouth. The teeth were extracted and the patient was advised to stop using tobacco in any form. He was seen periodically every two months until April, 1943, during which time the leucoplakia had remained the same.

Comment.—Leucoplakia is a precancerous lesion and if allowed to persist, it may thicken and fissure. As long as it remains soft and pliable to touch, there is no indication for instituting active cancer treatment. But, any area of leucoplakia which is even slightly indurated should be biopsied. The treatment of

choice, usually, is close observation with improvement of oral hygiene. In this case, the teeth were extracted and the tobacco-chewing habit stopped, and the lesion remained unchanged for two years.

Fig. 8.—Case 6. Leucoplakia.

Fig. 9.—Case 7. Chronic inflammation.

Case 7.—D. G., a 64-year-old white male, referred by his physician, was first seen in clinic in October, 1941, complaining of a growth on the hard palate. The patient stated that about two years ago, while he was eating, a fish bone became lodged between the gum and the upper right second molar. Shortly afterward, he noticed a small growth in this area which had grown gradually ever since. Examination of the mouth revealed a small, nodular, red growth measuring 1 by 1.5 cm. on the right side of the hard palate opposite and next to the first and second upper molars (Fig. 9). It was soft to touch, nontender, and did not bleed. The remaining teeth were in very poor repair. Pathologic examination showed only chronic inflammation. Biopsy excision of the right upper second bicuspid, first and second molars with the granulomatous tissue adhering to the roots and the hard palate, showed chronic inflammation, fibrosis, and sclerosis. Recovery was uneventful. The case was closed after fourteen months of observation with no evidence of recurrence.

Comment.—This case well illustrates the lack of oral hygiene. The lesion noted on the palate was an extension of the granulomatous tissue present on the roots of the upper right posterior teeth. These growths are benign and should be widely excised to prevent recurrence.

Case 8.—D. S., a 35-year-old white female, referred by her physician, was first seen in the clinic in February, 1942. The chief complaint was a slowly growing mass on the upper gum over the right incisor space, of about two and a half years' duration (Fig. 10). The patient stated that she had had pyorrhea ever since she could remember, and that about two and a half or three years ago she noticed a small raised area at the interdental space between the upper central incisors. This slowly increased in size until about a year ago, when she consulted her dentist, who extracted her teeth and assured her that the growth would disappear. Her local dentist constructed an upper denture over the tumor mass in the anterior upper jaw. The lesion became more tender and the patient consulted a physician, who referred her to this hospital with question of cancer. A biopsy excision of the lesion on the anterior maxilla showed acute and chronic inflammation. The patient refused to return for follow-up, but word from her confirms no evidence of recurrence.

Fig. 10.—Case 8. Chronic inflammation which was present at the time a denture was made.

Comment.—The construction of a denture with a lesion remaining in the mouth is beyond all scientific reasoning. The fear or refusal of some dentists to refer patients to hospitals or seek consultation on questionable lesions is responsible for the high percentage of unrecognized, early malignant lesions. Fortunately, this lesion was benign and the patient suffered only mental distress.

Case 9.—N. B., a 16-year-old white female, referred by her physician, was first seen in the clinic in November, 1940. Her chief complaint was a growth between the upper cuspid and the first premolar, first noticed in 1938. It had been surgically removed by her local dentist twice, with a larger recurrence each time. Examination revealed a small lesion measuring 0.5 by 0.5 cm. between the upper right cuspid and premolar, and a loss of contact between these teeth (Fig. 11). The patient complained of food impaction at this interdental space, a condition which she said had existed as long as she could remember. A

biopsy excision was done which showed inflammation. She was referred to her home town dentist for the construction of an inlay restoration to establish contact between the cuspid and first premolar. The patient was examined again in December, 1940, with the inlay restoration in place, which, incidentally, was not the type indicated, and there was evidence of recurrence of the growth. This lesion was excised and the area was treated with 780 r units of x-ray therapy. Examination in September, 1941, revealed a much larger lesion than before. The patient was admitted, the inlay removed, and the lesion was again surgically excised and an inlay of our own design was placed into the premolar. The patient was last seen in October, 1942, at which time there was no evidence of recurrence (Fig. 12).

Comment.—This case illustrates how a granuloma will tend to recur if the irritation is not removed.

Fig. 11.—Case 9. Granuloma. This had been excised twice with a larger recurrence each time.

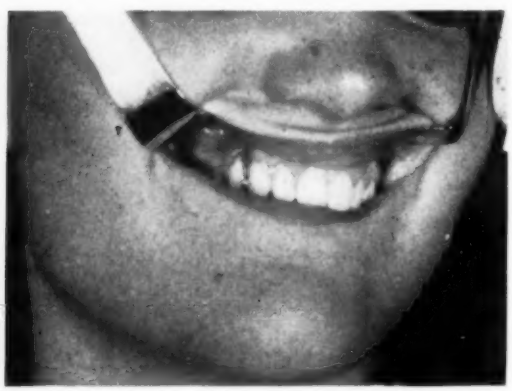

Fig. 12.—Case 9. One year following excision and restoration of contact.

CASE 10.—J. L., a 78-year-old white male, referred by a dentist, was first seen in the clinic in May, 1942. His dentist had noticed an area of hyperplastic tissue on the lower anterior gum and refused to extract the teeth as the patient had requested. This was the first the patient knew of any growth in his mouth. Examination revealed a hyperplastic area on the labial portion of the right lower anterior gum (Fig. 13). Biopsy report showed hyperkeratinization,

hyperplasia, and chronic inflammation. Another biopsy, taken two days later, was similar. A third biopsy was done, and the microscopic report was "Marked prolongation of the rete pegs with a few areas in the deeper portion of the section which might represent an extremely well-differentiated epidermoid carcinoma. However, the biopsy has not been taken deeply enough to determine whether or not there is actual invasion present, and I cannot make a diagnosis of carcinoma from these sections with a clear conscience. The report still stands hyperkeratinization, hyperplasia, and chronic inflammation."

Fig. 13.—Case 10. Chronic inflammation of the lower gum.

X-ray report showed the alveolar margin on the right to be slightly irregular, both in topography and density, with resorption noted about the roots of the remaining mandibular teeth. All the remaining teeth were extracted and radiotherapy was given to the lesion in the anterior lower jaw. The patient was last seen in January, 1943, at which time there was no evidence of recurrent disease, no palpable cervical adenopathy, and the entire lower gum was devoid of any hyperplastic changes.

Comment.—This was the one case out of the ten studied which was first recognized and referred by a dentist. This proved to be nothing but an inflammatory lesion, but the home town dentist displayed intelligence by requesting a consultation.

SUMMARY

An attempt has been made through the presentation of cases to demonstrate the dentist's responsibility in the diagnosis, prevention, and treatment of precancerous and cancerous conditions of the oral cavity.

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THE MAXILLARY SINUS AND DENTAL PROCEDURES

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MANY dental operations upon the teeth and tissues of the upper jaw bring us close to the maxillary sinuses. The dentist is therefore obliged to bear in mind the need for safeguarding this area from direct or indirect invasion and infection as a result of any dental manipulation.

Improper and partial procedures in root canal therapy often yield no exciting symptoms of inflammation to compel the patient's attention to seek treatment. Very often, the patient is unaware that any untoward condition is taking place, nevertheless, a subacute or chronic process may be established to the great detriment of the patient's health and safety. Many such involved antra go unnoticed for long periods. They do not draw the attention that an acute condition does, but they are treacherous and a menace, nonetheless. The dentist must, therefore, do everything to keep them normal and sound, as well as to discover diseased antra and advise their treatment.

The following data relative to the subject under consideration should aid the dentist, whether young or old, in his daily practice.

Anatomy.—The maxillary sinuses (antra of Highmore) are paired cavities occupying the interior of the maxillary bones and conforming, in the main, to their shape. The fossa of the sinus is of a quadrangular pyramidal shape, with its base lying mesially and its apex laterally toward the zygomatic or malar bone. The mesial or nasal wall communicates with the middle meatus of the nose by a small aperture, the ostium maxillare. The antero-external wall forms part of the face; the superior wall forms the floor of the orbital cavity; the posterior wall articulates with the sphenoid bone, and the inferior wall helps to form the hard palate of the oral cavity. The capacity of the sinus varies from 2 drams to 1 ounce. All the paranasal sinuses are lined by a mucoperiosteum, which is continuous with the mucous membrane of the lacrimal sac and pharynx. This continuity is important because of the interrelationship of diseases of these areas.

Etiology of Maxillary Sinus Disease.—The paranasal sinuses are subject to inflammatory changes that take place during certain infectious diseases such as severe colds, influenza, and scarlet fever. The maxillary sinus may become involved secondary to infection of the ethmoid or frontal sinuses. Dental pathologies as well as certain dental procedures and manipulations may be the etiological factor, to wit: pulp degeneration and suppuration, and abscesses of any of the dental units of the superior maxilla from the cuspid to and including the second molar. The antrum may be involved either by direct contamination, which takes place when a broach or probe is passed through the apical foramen into the adjacent tissues or directly into the antrum, in instances where the roots of the tooth involved protrude directly into the antrum, or when a root or roots of an infected or abscessed tooth is accidentally forced into the antrum during extraction. Also, it may be involved as a

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result of disintegration and suppuration taking place in the enamel organ of impacted teeth as well as in the surrounding tissues. Other causes are: extension of periodontal disease, the cumulative effect of residual infection from extracted teeth, and infection and involvement from drainage of the other paranasal sinuses communicating with the antrum.

Pathologic Findings.—The pathologic changes which occur in the mucous membrane and bony walls of the sinuses during the course of an infection are the same as would occur in any mucus-lined cavity. These changes may be described as acute congestive, acute suppurative, chronic suppurative, and chronic hyperplastic. Polyps, which may occur in any of the sinuses, are more frequently found in the maxillary and ethmoidal sinuses. Ulceration of the mucosa with involvement of the underlying bone takes place particularly in the chronic form of sinusitis. Bacterial cultures in chronic maxillary sinusitis most often yield *Staphylococcus albus* and *Streptococcus viridans (nonhemolyticus).*

Signs and Symptoms.—In *chronic sinusitis* there is usually an absence of pain; however, there may be occasional headaches, which do not refer to the affected side except during an acute exacerbation. Tenderness and pain upon pressure over the thin antero-external wall of the sinus is usually present. The amount of discharge through the nose is slight, or entirely absent; but there may, however, be a postnasal discharge. Inflammatory enlargement of the regional lymph nodes is frequently seen.

In *acute maxillary sinusitis*, pain and headache referable to the region of the involved sinus are present. In acute ethmoiditis there is a sense of fullness or pressure between and above the eyes. If the sphenoid sinus is involved, the headache may be vaguely deep-seated in the head, or the pain may be referred to the occipital or frontal regions. In acute sinusitis the formation of pus is ushered in with a rigor, followed by a rise in temperature and general systemic disturbances. Generally, there is an offensive, purulent discharge from the nostril of the affected side if the normal opening into the nose is open. When due to induration of the lining mucous membrane, the ostium maxillare is closed, the pain is then very intense, shooting over the face, forehead, and ear. Patients are very likely to attribute the pain to the teeth on that side. In the presence of a dental defect, it is often difficult to make a differential diagnosis and even more difficult to convince the patient that it is not caused by a tooth that you may have treated. Protrusion of the antral wall and, later, absorption of the thin lamina may take place; rupture through the soft tissues occurs, and relief is experienced. In rarer conditions, the pus may burrow posteriorly, after having penetrated the floor of the orbit, and may enter the brain through the sphenoidal fissure or the optic foramen.

The discharged material is of a highly foul odor. Giddiness and vertigo, or blurred vision and a feeling of faintness may be complained of. The appetite is poor, mental vigor and interest are lessened, and fatigue comes on readily. The blood count usually shows a moderate leucocytosis.

Routine Examination.—From the dental angle, the examination must be complete and thorough in order to make a definite determination. All related teeth must be suspected and roentgen-rayed so as to show all of the periapical

area from tooth apex to and including the floor of the sinus. Teeth apparently vital, but giving either a hyper- or a hyporeaction to thermal test or galvanic current, must be suspected, on the ground that we know that the pulp of a tooth may be infected before we recognize clinically an exposure. The clinical history and appearance of the tooth must be taken under consideration in order to determine its degree of involvement, for we must constantly bear in mind that type of low-grade infection, which, while symptomless, is far from innocent. The presence or absence of any unerupted supernumerary or malposed or impacted teeth, as well as of any foreign body, must be determined. Transillumination and x-ray of the sinuses are further clinical aids, though, if negative, they are not necessarily conclusive. Transillumination is definite if the sinus contains fluids, or a thickened mucoperiosteum, or large polyps. Posteroanterior x-ray plates of the sinuses should be taken with the jaws slightly apart so as to allow for a clear visualization of all the paranasal sinuses including the sphenoidal. The roentgenologist may report the plates as being negative, meaning, thereby, merely that there is no fluid, polyp, neoplasm, or foreign body present, but not that it is necessarily a normal antrum.

It is necessary, therefore, to assume that the presence of any periapical, or periodontal pathosis is sufficient reason, especially when taken together with the history of the case, to justify every effort to make an exhaustive search before a final diagnosis is made.

From the statements above, it must be clear that the plea is made solely for the purpose of a thorough diagnosis and is not to be construed as urging the dentist in general practice to perform operative surgery on the sinuses. Often it will be found that infection of one sinus has led to involvement of one or all of the other paranasal sinuses. In such instances, the dentist or dental specialist may, by law, go so far only, after which he will have to seek the cooperation of the rhinologist.

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HISTOGENESIS OF THE SO-CALLED SECONDARY ENAMEL CUTICLE AND DENTAL CUTICLE

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THIS paper is concerned with the attempt to apply conclusions drawn from histologic findings in a pathologic condition to the interpretation of a structure occasionally found on the normal tooth. This method of interpretation is not at all unusual for it was often effectively used in histology to show the genetic relations between cells and tissues.

Since Gottlieb[1] proved the presence of a secondary enamel cuticle deposited by the epithelial attachment upon the primary cuticle (Nasmyth's membrane), this discovery has been accepted. Gottlieb[1] and Orban[2] contributed much to our knowledge of the secondary enamel cuticle by pointing out the basic etiologic difference between the primary and secondary enamel cuticle and the different reactions of these two structures to acids and alkalies. The resistance of the secondary enamel cuticle against acids and alkalies is thought to be a kind of barrier against dental caries. Both authors also stated that the deposition of the secondary enamel cuticle may go on as the epithelial attachment shifts along the cementum. They called this same product of the epithelial attachment, which occasionally and locally coats the cementum, the dental cuticle. They emphasized that this cuticle is not always present on every tooth and, if present, the band is not continuous.

While there is full agreement between the investigators as to the existence of a secondary enamel cuticle and dental cuticle, there is disagreement as to the genesis and nature of this structure. Concerning the origin, Gottlieb[1] described this cuticle which appears bright pink, in hematoxylin- eosin stained sections, as a hornified layer of the epithelial attachment superimposed upon the tooth surface even after the recession of the epithelial attachment. This conception was supported by Orban,[2] Häupl,[3] and Bauer,[4] whereas, among others, Lehner[5] recently considered the secondary enamel cuticle, and consequently also the dental cuticle, as a surface secretion of the epithelial attachment, in other words, as a true structureless cuticle of homogenous appearance in the strict histologic sense. Both groups of investigators agreed that the dental cuticle in sections only occasionally stains with dyes that serve to demonstrate horny substances. I tried to interpret this fact in my former study[4] by conceiving of the dental cuticle as a structure of keratohyalin that might become cornified. I also claimed and demonstrated that this structure may occasionally show, more particularly in the beginning of its formation, inclusion of cells and granules. My findings led me to conclude that the dental cuticle is due to a transformation of the epithelial layer adjacent to the tooth into a kind of a hyalinized membrane that may or may not become cornified. In disagreement with my conception which supports Gottlieb[1] and Orban's[2] idea, Lehner[5] refused to

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consider this structure as a horny epithelial product and designated it as a true cuticle, a stable secretion of the uppermost epithelial layer. Furthermore, Lehner[5] classed these "cuticles" under the varieties of basal epithelial membranes that attach the epithelium to the connective tissue. However, he emphasized the importance of further studies to determine the reason for the irregularity of its occurrence.

MATERIAL AND METHODS

The observation of a "secondary cuticle" on the enamel of a completely embedded upper third molar, lying above the decayed root-rests of the second molar is the main subject of this report. The specimen was obtained and necropsy performed on a man of 38 years who died from tuberculosis. Another specimen, also removed at necropsy, contained the upper second molar with all supporting structures and the floor of the maxillary sinus. I believe that the unusual findings in these cases throw some light on the controversial problem of the secondary enamel and dental cuticle.

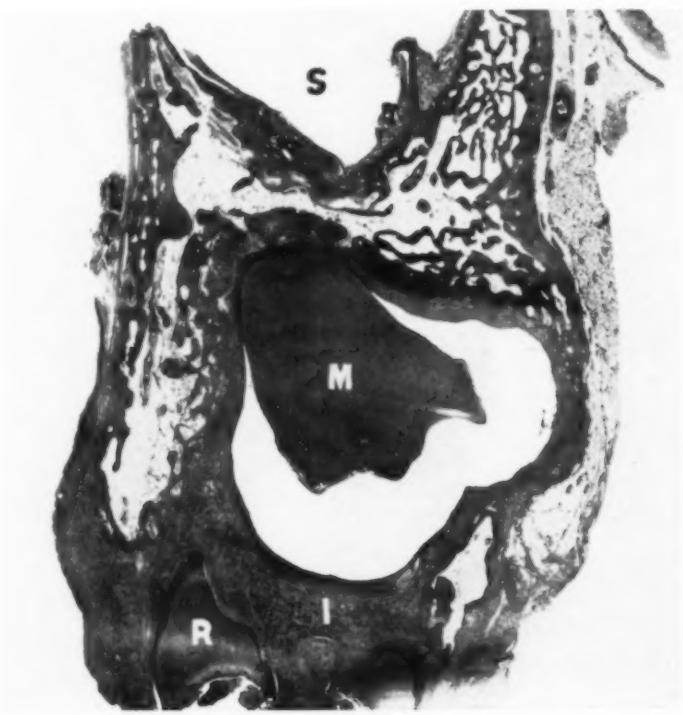

Fig. 1.—Buccolingual section through an embedded upper third molar (*M*) and the remnants of the proximal buccal and palatal roots (*R*) of the second molar. *I*, Chronic inflammation with epithelial strands proliferating from the root ends to the enamel epithelium of the embedded tooth; *S*, maxillary sinus with mucosa. Hematoxylin-eosin stain.

Celloidin sections of this material were stained with hematoxylin-eosin, with Mallory's aniline blue for hyaline and horny substances, and also by the Buzzi method modified by Unna to differentiate between eleidin and keratohyalin. In order to use Mallory's aniline blue method, the sections cut from a formalin fixed specimen embedded in celloidin were placed in a saturated solution of corrosive sublimate for nineteen hours, after which time they stained brilliantly.

MICROSCOPIC OBSERVATIONS

The buccolingual sections of the first specimen (Fig. 1) contained the remnants of the proximal buccal and palatal roots of the upper second molar above which the fully embedded third molar was located. The epithelial attachment on the cementum of the roots, which had no alveolar socket and lay in connective tissue, proliferated along the cementum layer forming projections into the inflamed adjacent connective tissue. The strands continued around the apical ends as massive accumulations and islands of epithelial cells within a fibrous granulation tissue containing lymphocytes and, mainly, plasma cells. Giant cells and cholesterol slits were scattered over the lateral parts of this granulation tissue area that completely surrounded the crown of the embedded tooth.

Fig. 2.—The histologic pattern of the highly inflamed tissue between the root end (*R*) and the enamel (*E*) of the embedded molar. Showing bright pink-stained bodies of hyaline nature within the broad band of proliferated epithelium. Hematoxylin-eosin stain.

This broad bandlike dense mass of epithelial cells, above the root apices, extended without interruption up to the reduced epithelial layer which covered the enamel of the embedded molar up to the cemento-enamel junction. It consisted of three to five rows of flattened epithelial cells. In other words, there

was a continuity of the epithelium encircling the roots with the reduced epithelium layer of the embedded tooth. The continuity was brought about by this broad band of proliferated epithelium.

Fig. 3.—High magnification showing the epithelial hyaline membrane (*M*) ("secondary enamel cuticle") of the embedded tooth. *C*, Primary enamel cuticle; *EP*, enamel epithelium of the embedded tooth; *I*, chronic inflammation; *E*, enamel of the embedded tooth. Hematoxylin-eosin stain.

The most outstanding findings essential to this paper were detected within this area of epithelial cell accumulation. There were bands, rods, rings, and spherically shaped bodies of an almost homogenous substance that appeared bright pink in hematoxylin-eosin sections, orange-red by Mallory's aniline blue, and bright yellow by Buzzi's method. The thicker bodies showed an inner core of deeply blue stained material in hematoxylin-eosin sections. These hematoxylin stained inner areas with faint outlines were structureless and could be easily distinguished from sharply limited nuclear remnants occasionally found within the brightly eosin-stained outer layers. The site of these bodies was strictly limited to the field of densely packed epithelial cells. These quaint structures reached to the enamel epithelium of the embedded tooth and continued here as a pink, or orange-red, or yellow-stained, more or less homogenous band, 4 to 10 μ thick (Fig. 2). This band, occupying the innermost layer of the enamel epithelium and encircling the crown, was almost continuous. It was interrupted rather infrequently by the appearance of the original epithelial cells and ended at the cemento-enamel junction. A dense small cell infiltration accompanied the enamel epithelium throughout (Fig. 3). This membrane was closely adapted to the enamel surface (Fig. 4), also outlining the fissures (Fig. 5), and reaching to an enamel lamella (Fig. 5). It appeared superimposed upon the primary cuticle (Fig. 3). The enamel epithelial layers were either in intimate contact with the membrane, as was most often the case, or they were occasionally separated from it by exudate. This enamel covering membrane was largely structureless, however, and tracing its course one could easily ob-

serve that it continued in a few places into the innermost layer of the enamel epithelium. This area of transition revealed remarkable changes: the bright eosin, or the orange-red, or the yellow stain gradually disappeared and an increasing number of nuclear remnants became noticeable.

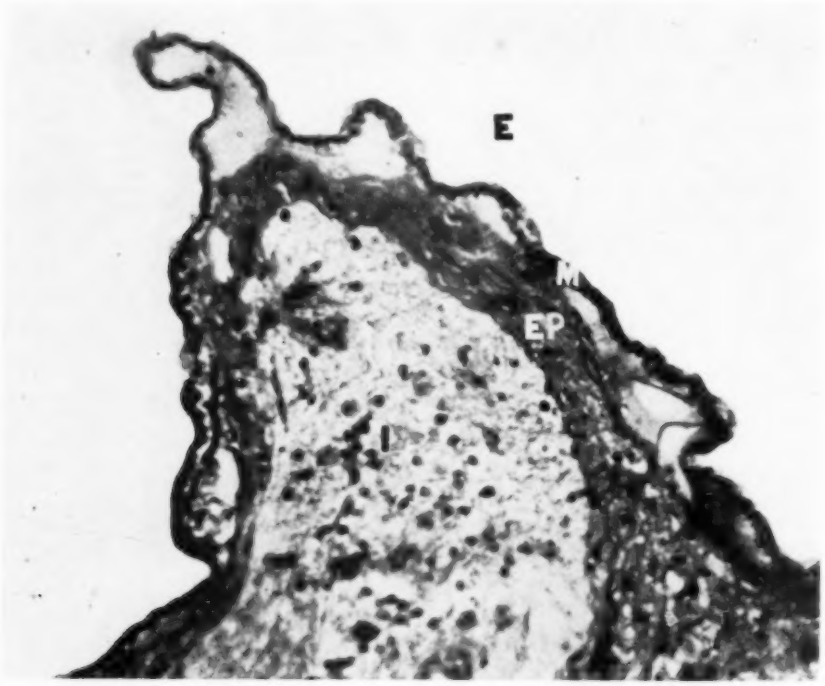

Fig. 4.—Mallory's aniline blue stain showing the horny epithelial hyaline membrane (*M*) ("secondary enamel cuticle") of the embedded tooth. *E*, Enamel; *EP*, enamel epithelium continuing into proliferated strands; *I*, small cell infiltration.

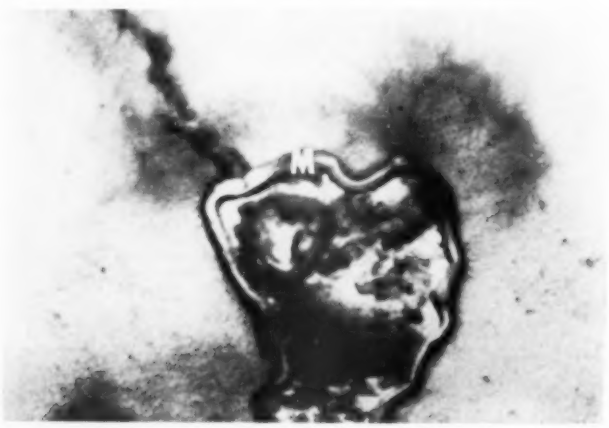

Fig. 5.—Mallory's aniline blue stain. Horny epithelial hyaline membrane (*M*) ("secondary enamel cuticle") in a fissure of the embedded tooth connected with an enamel lamella.

The most striking microscopic finding was the evidence that there was a continuity of the "dental cuticle" surrounding the cementum of the decayed roots with the structureless masses within the epithelial strands between these roots and the embedded tooth, and also with the membrane described which extended along the enamel of the latter and covered its primary cuticle. These

three structures, connected with each other, exhibited the same affinity to eosin stain and also to differential stains.

The sections of the second specimen showed an enamel drop in the bifurcation of an upper second molar coated by a broad band of squamous epithelium. This proliferation of the epithelium resulted from the severe inflammatory infiltration of the subepithelial tissue between bone and tooth. The epithelial attachment shifted apically and thus exposed the bifurcation. Proliferating epithelium grew over the surface of the enamel pearl after inflammation had destroyed both the periodontal membrane and the interradicular alveolar crest in this area. The routine hematoxylin-eosin stain revealed the presence of a homogenous membrane, about 7 μ thick, between the enamel pearl surface and the thick epithelium. This bright pink-stained membrane not only covered the enamel surface but also branched into the epithelium, turning some of its cells into eosin-stained islands and continued on as a typical "dental cuticle" along the cementum of the roots. Mallory's aniline blue stained the membrane and its extensions red, while Buzzi's method produced a yellow stain.

DISCUSSION

In order to obtain a fairly clear picture of the relation existing between these structures in question and the epithelium, one must study the early formative stages of these products. The investigators, particularly those who emphasized the cuticle nature of the "secondary enamel cuticle" and the "dental cuticle" and denied their horny nature, did not have the opportunity to study the developmental stages, for they observed the already developed cuticles adjacent to an unaltered epithelium. The presence of columnar epithelium bordering the "secondary enamel cuticle" induced Lehner[5] to refuse the conception that this membrane is a horny layer. It is true that the epithelial cells adjacent to the fully developed "epithelial membrane" in the cases reported appeared to be unchanged. They showed neither flattening, nor shrinking, nor keratohyaline granules. However, the epithelial cells in intimate connection with the developing "epithelial membrane," wherever found, presented instructive changes. Their cytoplasm increased remarkably in volume, the nuclei became somewhat enlarged and faint, appeared reticulated, and diffused into the cytoplasm while the outlines of the cells disappeared so that their cytoplasm, still stained pale blue, coalesced (Fig. 6). Threads of cell membranes left behind were slightly evident in these bands of coalesced cytoplasm that, arranged in layers, gradually became homogeneous and acidophilic. The cytoplasm appeared bright pink in sections stained with hematoxylin-eosin, bright orange-red with Mallory's aniline blue, and bright yellow by Buzzi's method. The fully developed epithelial membrane finally appeared, bordered by normal flat epithelial cells.

On the basis of these findings it seemed that all these membranes, being of epithelial origin, were a product of a transformation of epithelial cells into an homogenous substance that coated the enamel of both cases as a membrane and also intervened, as a so-called "dental cuticle," between the cementum and the epithelial attachment. They could not be regarded as a secretion product as is true cuticle. Though being fully aware of the shortcomings of determining the nature of a substance by selective stains, I conclude, from the reaction to

various stains but particularly from the study of their developmental stages, that these membranes were of horny nature in spite of the fact that there was no evidence of keratohyaline granules either in the epithelial cells or in the membranes. This conclusion is supported by the fact that keratinization occurs also without keratohyaline granules under abnormal conditions. That keratohyaline granules are present in the stratum granulosum of the gingiva under normal conditions was proved by Orban[6] and Weinmann.[7]

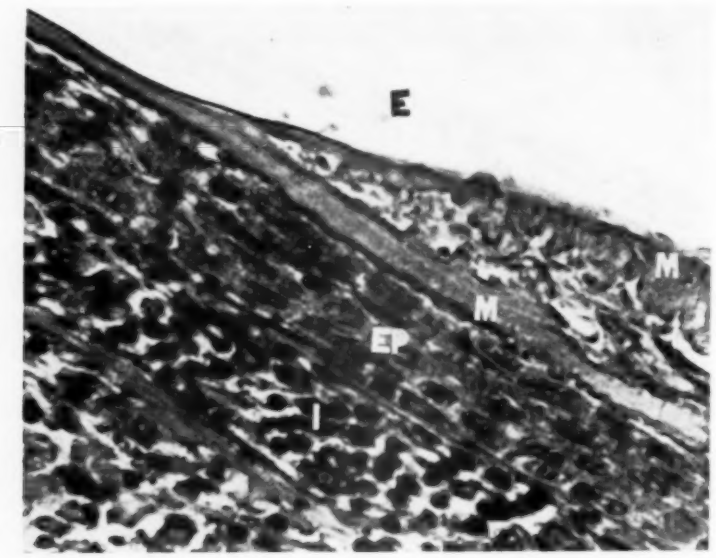

Fig. 6.—Formation of the hyaline membrane (*M*) by the enamel epithelium (*EP*) of the embedded tooth. Note the changes of the epithelial cells lying between the two branches of the hyaline membrane (*M*) which unite at the left; *I*, chronic inflammation.

The detailed study of the process of formation of the horny bodies and membranes in question, however, justified the opinion that a transformation of epithelial cells into a homogenous substance, as described, preceded the keratinization. I consider the original homogenous bands to be of a substance at least related to, and probably identical with, epithelial hyalin. This epithelial hyalin may or may not become keratinized. In the cases reported, most of it appeared to be horny. Hornification of epithelial hyalin is not at all uncommon under inflammatory conditions and the keratinization of the epithelial bodies in squamous cell carcinoma is an outstanding instance of this. The "secondary enamel cuticle" and "dental cuticle" in sections of other human specimens and also the epithelial pearls in squamous cell carcinoma showed the same affinity to the differential stains employed as the membranes in the cases presented. There could not be any doubt that the horny epithelial hyaline membrane covering the enamel of the embedded tooth and the enamel pearl represented what is called the "secondary enamel cuticle." Furthermore, the development and the nature of these horny epithelial hyaline-like membranes were identical with the development and the nature of the rods, loops, and islands of horny hyalin within the densely packed epithelium that reached from the apices of the decayed molar roots to the embedded molar and were also observed in the epithelium extending along the enamel pearl.

The routine hematoxylin-eosin stain and Mallory's and Buzzi's methods demonstrated, occasionally, the absence of cornified areas within the hyaline-like membranes, thus explaining why some investigators found the "secondary enamel cuticle" and the "dental cuticle" to be of hornified substance, while others did not.

Finally, the question as to the relation existing between the epithelial hyaline-like membranes in the cases presented and chronic inflammation is to be answered. It is in this connection that a comparison of these cases with former studies[8] I conducted seems to be appropriate. The latter were concerned with the pathology of the so-called "Turner teeth." I proved on autopsy material that a flare-up of a chronic periapical infection of deciduous teeth resulted in destruction of the enamel epithelium of the underlying buds of the permanent teeth, in the resorption of their enamel and also replacement of it by a bone-cementum.

Contrary to this destructive effect, the severe chronic inflammation observed in the cases reported in this paper resulted in proliferation of the enamel epithelium and formation of horny epithelial-hyaline-like membranes closely adapted to the enamel surface. Although no intelligent explanation of this contrary reaction can be given, one cannot help considering these structures as a product of chronic inflammation. Keratinization and hyaline formation have been generally regarded in the past as degenerative processes, but the modern conception tends to regard them also as a protective reaction. I strongly believe that the findings reported in this paper support this idea. The conception of the possible inflammatory origin of the horny epithelial hyaline-like membrane may throw some light on the irregularity of its occurrence. Since the "secondary enamel cuticle" is considered a product of the epithelial attachment formed during tooth eruption, and the "dental cuticle" is regarded as a product of the epithelial attachment during its migration along the cementum, and both of these processes are usually associated with inflammatory changes in the subepithelial connective tissue, the possibility exists that the presence or absence of these hornified membranes might depend on the grade of the inflammatory process.

SUMMARY

A "secondary enamel cuticle" was observed on the enamel of an embedded tooth and also on an enamel pearl in the bifurcation of an upper molar.

These "secondary enamel cuticles" continued into horny hyaline-like bodies within densely proliferated epithelial strands which were a product of chronic inflammation. In the first case they extended from the embedded third molar to the decayed roots of the second molar, and in the second case they covered the enamel pearl in the bifurcation of an upper molar.

The so-called "secondary enamel cuticle" and "dental cuticle" are not a secretion product of epithelial cells but a horny epithelial hyaline-like membrane.

The formation of this horny epithelial-hyaline-like membrane seems to be in a causal relation to chronic inflammation which would satisfactorily explain the occasional occurrence of the membrane.

The suggestion is made to discard the terms "secondary enamel cuticle" and "dental cuticle" and to reserve the name "*horny epithelial hyaline-like membrane*" for both these structures. This term is compatible with the histologic and morphologic appearance of this membrane and clearly points out the fundamental difference existing between this membrane and the primary enamel cuticle.

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Case Report

CASE NO. 86

ADAMANTINOMA

MATTHEW LOZIER, B.S., D.D.S., NEW YORK, N. Y.

THE patient, a 37-year-old married white woman, born in the United States, a clerk by profession, was referred to the office on Sept. 13, 1943. She related the following history:

"At the end of April of this year, I noticed a slight lump in the rear of my mouth on the inside of the left cheek where the cheek joins the gum. I had absolutely no pain and felt otherwise fine except for some rheumatic pains in my left arm. Thinking that my rheumatic pain might have something to do with my jaw, I consulted a dentist, who, after examining my mouth, told me to 'forget it, unless the swelling begins to pain you.' A few days ago, about five months after I first consulted the dentist, I thought that the swelling had become a bit larger and heavier, and though I still had absolutely no pain, I went for advice to another dentist, who after examining me and taking a small x-ray of my jaw, told me that I had a cyst, which, because of the small size of the x-ray, did not show in full. I was then referred to you for an operation on the cyst."

Further questioning revealed the information that about twelve years ago a "cyst attached to the roots of the left lower impacted molar" was removed for the patient in one of the large new York hospitals. The operation was quite difficult and lasted over an hour. The patient added that she was then also aware of a "pronounced cordlike swelling extending from the angle of the left jaw down on the neck," which disappeared soon after the operation. The patient did not remember being recalled for postoperative check-up. Our communication with the above institution for a history of the case brought no reply.

Examination.—The patient appeared to be an intelligent, well-nourished woman with no apparent systemic abnormalities, with a normal past and family history.

Extraorally, there were absolutely no signs of disfigurement. Intraorally, there was evidence of a slight swelling, soft in character and somewhat tender to the touch, over the mucobuccal fold at the left lower molar and retromolar regions extending over the anterior portion of the ascending ramus. There was no evidence of fluctuation and the lymph nodes were not palpable. The right side of the jaw was normal.

Roentgen Examination.—The intraoral radiographs taken periapically and occlusally, and positioned over the left molar and retromolar regions as far back as possible, disclosed a portion of an extensive radiolucent area with numerous

irregular islands or partitions of somewhat denser consistency. The occlusal view clearly indicated the involvement in parts of both the alveolar plates (Figs. 1 and 2). The extraoral view showed a large clearly defined circumscribed lesion involving the molar and retromolar regions, together with a portion of the anterior aspect of the ascending ramus. A part of the mandibular canal was, apparently, also encroached upon. The general appearance of this roentgenographic view was that of a typical adamantinoma (Fig. 3).

Fig. 1. Fig. 2.

Fig. 3.

Because the possibility that a complete or partial resection was indicated, if this tentative diagnosis was found to be correct, an impression of the jaw was taken before the biopsy examination, in the event a splint would be needed after the operation.

Examination of Tissue After Biopsy. (New York Institute of Clinical Oral Pathology.)—Gross examination disclosed two particles of tissue which were received in a fixative. One measured 7 by 5 by 1 mm., the other, which was irregular in shape, measured 2 by 2½ by 1 mm. A small amount of blood clot was received with the specimen.

Microscopic examination revealed that the greatest portion of the section was blood clot. However, there was a small island of tumor tissue present. This consisted of fairly dense connective tissue with epithelial cell nests. The outer border of these nests were formed by high columnar epithelium; the center by loose reticular epithelium. Occasionally, the lumen was empty, indicating the beginning of cyst formation. In one section there was some suggestion that the tumor was connected with a bony fragment, but it could not be definitely stated that the tumor had infiltrated the bone, nor did another small fragment of bone show any infiltration by the tumor. A smear of the fluid showed only debris; no definite microorganisms could be seen.

Diagnosis.—Ameloblastoma. Another examination performed at the Memorial Hospital confirmed the diagnosis of ameloblastoma.

On October 25 a complete resection of the left lower jaw was made, when the left submaxillary gland was also found to be involved.

DISCUSSION

I consider the case to be of interest for several reasons, which are as follows:

1. Although the patient underwent, some years ago, an apparently complicated operation with some glandular involvement, she was never recalled for a re-examination, nor was she informed as to the need of a periodic recheck. If this had been instituted, earlier discovery of the present lesion would have been possible, and a resection, partial or complete, possibly avoided. The fact remains that even the present discovery of the condition was purely accidental.

2. The advice of the dentist to whom the patient first came for a consultation, to "forget it, unless it starts paining" indicates the necessity for more careful clinical and particularly roentgenographic examinations of all cases regardless as to how innocent they may appear.

3. Tentative diagnosis, with roentgenographic and pathologic evidence incomplete, should be always guarded. Extraoral roentgenograms are indicated in all jaw lesions in order to disclose the complete area of pathosis.

4. The employment of a biopsy examination before rendering a final diagnosis, and before instituting surgery, is necessary in all cases. This should be done regardless of the intention of having the removed tissue examined after the operation.

5. When dealing with a condition where the surgical procedure may require a complete or partial resection, or where a fracture is anticipated, a cast should be made of the patient's jaw before the operation. This saves a good deal of discomfort to the patient and offers the surgeon a more accurate model for the construction of a splint.

369 East 149th Street

Editorial

Vincent's Infection in the South Seas

Major C. S. Linton, M.C., A.U.S., makes a preliminary report* on the use of sulfathiazole locally applied. He states that Vincent's infection of the tonsils was seen recently in several soldiers returning from the South Seas. These men had been exposed to strenuous physical conditions for a considerable period of time with meager rations while on front line duty. They were all sent back to the zone of the interior with various types of disability. They were treated with sulfathiazole in the following manner: A 0.5 Gm. sulfathiazole tablet was administered every two hours to be dissolved on the tongue during daytime, and 1 Gm. was similarly given every four hours at night. This treatment gave relief in two days, and then it was discontinued unless directed otherwise. When the infection was also present around the gingival margins, it was advised that the sulfathiazole tablet be moistened with a few drops of water to make a paste, which was used to run into the gingival crevices. While excellent results were secured in one case, the treatment of Vincent's infection of the mouth was not sufficiently tried to warrant any expression of an opinion. Four cases of Vincent's angina of the throat, remarkably relieved by the sulfathiazole treatment, are appended. Two of the patients had previously been treated with other methods without effective result (intravenous injection of neoarsphenamine in one, and mapharsen, combined with local application of sodium perborate paste to the gum margins, in another). The article is a preliminary report made at this time on account of the likelihood of pronounced increase of cases of this type under war conditions. The writer takes into consideration that some associated infection may prepare the ground, and that nutritional factors may play a part. He believes that recurrences of the infection are likely, no matter what the treatment, unless factors which cause weakened resistance in the tissue are controlled.

K. H. T.

*Linton, C. S.: Treatment of Vincent's Angina of the Tonsil, J. A. M. A. 123: 341, 1943.

Abstracts and Reviews

Applied Anatomy of the Head and Neck: By Harry H. Shapiro, Assistant Professor of Anatomy, College of Physicians and Surgeons, Columbia University, Pp. 178, Price $5.50, Philadelphia, J. B. Lippincott Co.

The author states that this book is for students and practitioners of dentistry. The anatomical basis for many corrective procedures in dentistry forces one to say that this book is a textbook of dentistry. At least, one has the desire to say that the information collected in this book is absolutely necessary for the practice of dentistry. It is a noval treatise on the anatomy of dental and related structures. Every discussion has practical value. There are some outstanding features in this book, one of which is the excellent attempt to demonstrate anatomy by means of x-ray films. Another outstanding effort is the discussion and demonstration of the temperomandibular articulation. To see such material in a single volume for students means that, finally, fundamental information for dental students is available in a single textbook rather than hidden some place in a collection of journals or reprints. Only one criticism may be offered, and that is that the oral surgeon who practices major oral surgery may find some details lacking; but the author states that this book is for students and practitioners of dentistry. This book will be useful to the medical man in many instances, if not all. The print is good, the pictures are excellent, and the index is very usable.

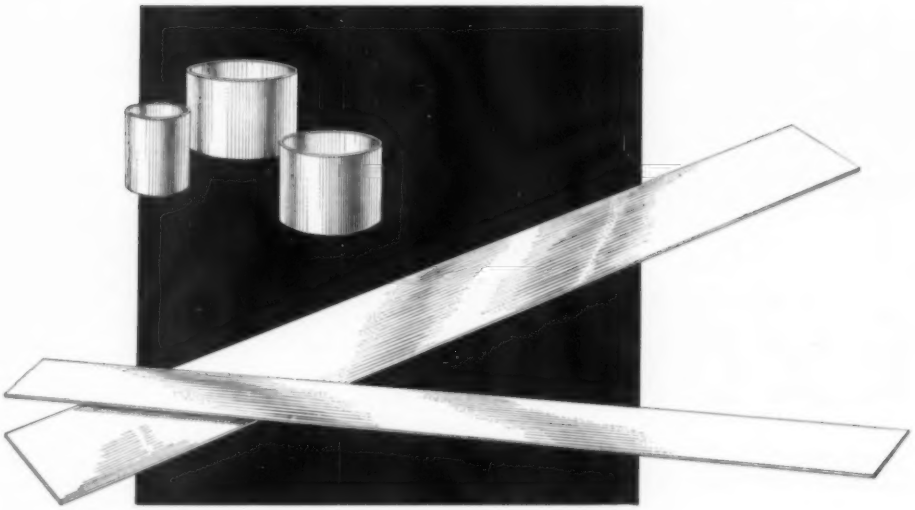
D. W.

The Dental Treatment of Maxillo-Facial Injury: By W. Kelsey Fry, L.R.C.P., L.D.S., R.C.S. (Eng.), Consulting Dental Surgeon to the Royal Air Force, Dental Surgeon to Guy's Hospital, P. Rae Shepherd, L.D.S., R.C.S. (Eng.), Dental Surgeon, East Grinstead Maxillo-Facial Unit, M.R.C.S., L.D.S., R.C.S., (Eng.), Pp. 246, Philadelphia and Montreal, J. B. Lippincott Co.

This book comprises the subject matter and demonstrations given to members of H. M. Forces at East Grinstead Maxillo-Facial Center. It is intended for those dental surgeons who have had little or no experience in the treatment of injuries about the face. There are fourteen chapters, which include fundamental anatomical considerations, clinical diagnosis, types of fracture, radiology, pathology, and complications. The chapters on reduction and fixation are condensed but are complete. Several pages are devoted to the use and, probably, the abuse of the Roger Anderson type of fixation. Practically every type of fixation ever thought of is demonstrated. Some new ideas relative to metal splints and splints made of other materials are included. The treatment of complications is occasionally at a variance with some newer opinions. The diagrams are clear, and the print is good. The attempt to reproduce x-ray nega-

tives could be improved, but this is a fault common to most similar efforts. In this book, although condensed, one can find almost any problem which can confront a dental surgeon. The solution as suggested in the column is the result of study by four dental surgeons, and this fact alone would make it a contribution to dental literature.

D. W.



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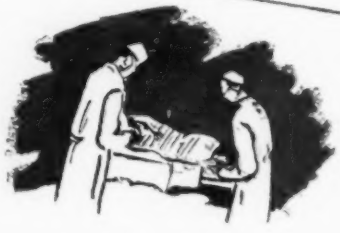

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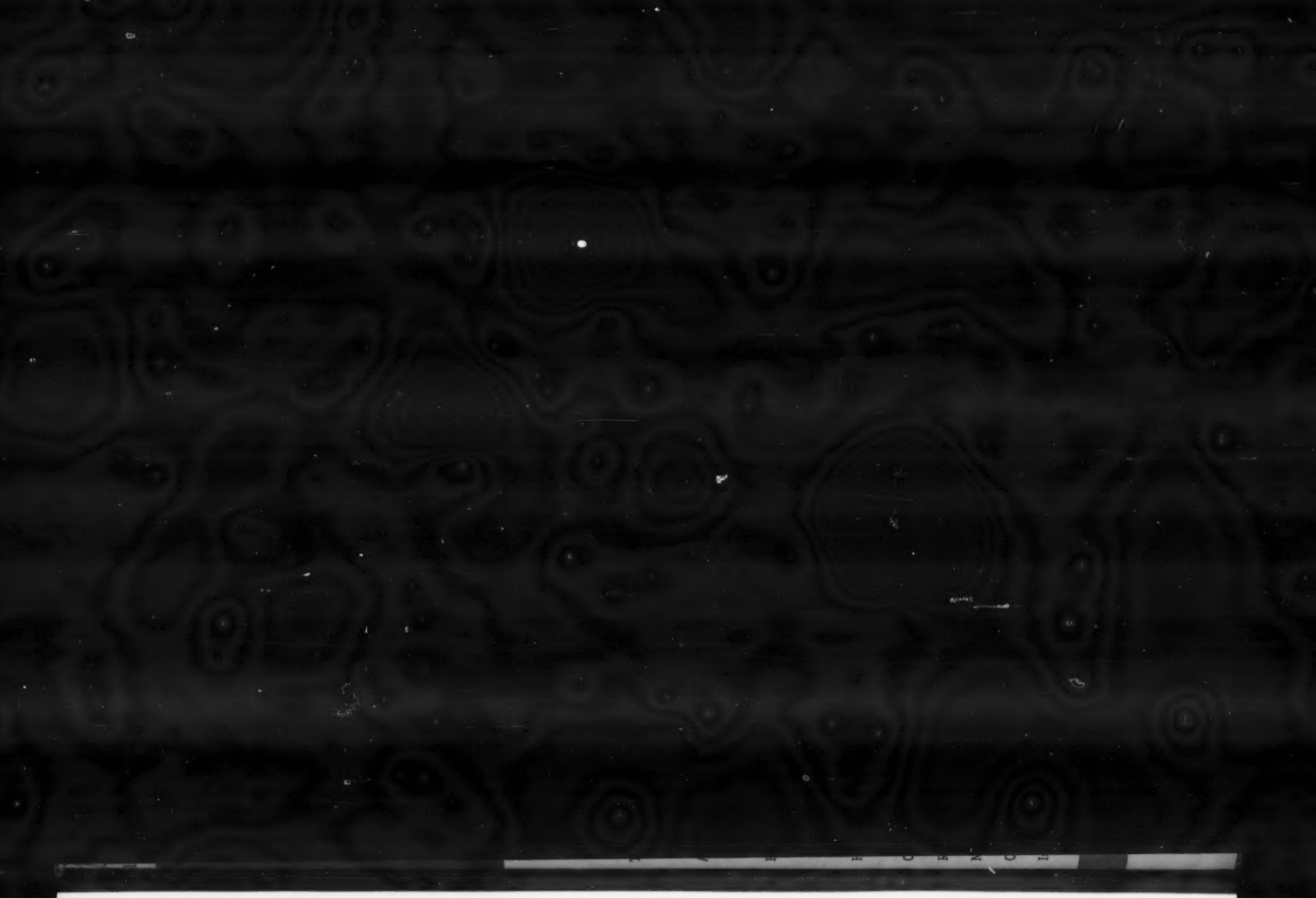
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